JAUNDICE COMPLICATING PREGNANCY – MATERNAL AND FETAL OUTCOME

Dissertation submitted to

THE TAMILNADU

Dr. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirement

For the award of

M.S.DEGREE - BRANCH – II

OBSTETRICS AND GYNAECOLOGY

Reg No: 221716014

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

MADRAS MEDICAL COLLEGE, CHENNAI.

MAY - 2020

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"JAUNDICE COMPLICATING PREGNANCY – MATERNAL AND FETAL OUTCOME"** is a bonafide record of original work done by **Dr. SARANYA. G** under the guidance of **Dr. GEETHA MAHADEVAN, M.D.,D.G.O.,** Professor of Obstetrics and Gynecology in Institute of Obstetrics and Gynecology, Chennai in partial fulfillment of the requirements for MS Degree in Obstetrics and Gynecology branch II examination of the Tamil Nadu Dr.MGR Medical university to be held in MAY 2020. The period of post graduate study and training from MAY 2017 to MAY 2020**.**

Prof. DR. SAMPATH KUMARI.S, M.D., D.G.O.,
Director I/c.,
Institute of Obstetrics and Gynecology,
Egmore,
Chennai.

Prof. DR. R. JAYANTHI, M.D., FRCP (Glasg),
Dean,
Madras Medical College
Rajiv Gandhi Government General Hospital
Chennai – 600 003

DECLARATION

I, Dr. SARANYA. G, Post Graduate, Department of Obstetrics and Gynaecology, Madras Medical College solemnly declare that this dissertation **"JAUNDICE COMPLICATING PREGNANCY – MATERNAL AND FETAL OUTCOME"** was prepared by me at Department of Obstetrics and Gynecology, Madras medical college, Chennai, under the guidance and supervision of Professor **Dr.GEETHA MAHADEVAN, M.D., D.G.O.,** Institute of Obstetrics and Gynecology, Chennai

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S. (Obstetrics and Gynaecology).**

Place: Chennai
Date:

(K.BUVANESHWARI)

Dr. GEETHA MAHADEVEAN, M.D.,D.G.O.
Professor
Institute of Obstetrics and Gynecology,
Egmore
Chennai

ACKNOWLEDGEMENT

I start my thesis in the name of almighty God. I thank him for giving me the privilege to learn from such eminent professors and assistant professors in my department. I express my sincere thanks to Professor **DR. JAYANTHI, M.D., FRCP (Glasg),** The Dean, Madras Medical College for allowing me to conduct the study using the available facilities.

I express my sincere gratitude to our Director Incharge **Prof.Dr.SAMPATH KUMARI S, M.D,D.G.O,** Institute of Obstetrics and Gynaecology, Egmore, Chennai for helping me in this study.

I convey my heartfelt gratitude and sincere thanks to my guide Professor **Dr. GEETHA MAHADEVAN, M.D.,D.G. O.,** Institute of Obstetrics and Gynecology, Egmore Chennai, who with her immense knowledge and professional expertise has provided guidance and encouragement throughout the study and in the preparation of this dissertation.

I thank all my professors,Assistant Professors of this Department of Obstetrics and Gynaecology,Madras Medical College,Chennai.

I thank all my patients for their cooperation and hence for the success of the study.I thank my family and friends for their inspiration and support given to me.

(SARANYA. G)

CONTENTS

`

INTRODUCTION

Jaundice in pregnancy, whilst relatively rare, has potentially serious consequences for maternal and fetal health. It can be caused by pregnancy or occur intercurrently. It is responsible for 10% of maternal deaths. The incidence of Jaundice in India varies from 0.4 to 0.9 per 1000 deliveries. Acute vital Hepatitis is the most common cause of Jaundice in pregnancy. Certain factors play an important role in the development of jaundice during pregnancy and in affecting the maternal and perinatal outcome. They are

1. Increased demand on the liver because the placental hormones are conjugated in the liver and produce changes in the liver.

2. CHANGES IN THE IMMUNE SYSTEM

   Increased production of progesterone during pregnancy leads to down regulation of cell mediated immunity. Many food pathogens are controlled by cell mediated immunity. So there is increased susceptibility to infections.

3. Associated hypo-proteinemia and anemia make the pregnant women more vulnerable.

Jaundice complicating pregnancy has been the cause for maternal mortality in 14% of maternal deaths during a 3-year study in the Institute of Obstetrics and Gynaecology, Government Hospital for Women and Children, Egmore, Chennai.

Therefore this study is undertaken with an aim to analyse the etiology, course of the disease and maternal and perinatal outcome in jaundice complicating pregnancy. It is sincerely hoped that this study will help in improving the maternal and perinatal outcome in jaundice complicating pregnancy.

Jaundice in pregnancy carries a grave prognosis for both the mother and the fetus, and is responsible for 10% of maternal deaths. Liver disease in pregnancy is an important medical disorder seen more often in developing countries than the developed ones. The present study analyzes the causes and the feto-maternal outcome in pregnancies affected with jaundice.

Abnormal liver test results are obtained in 3% to 5% of pregnancies because of many potential causes and the clinical outcomes ranges from self-limiting to rapidly fatal.

The main causes for abnormal liver tests in a pregnant patient are:

1. **Pregnancy-related liver disease.** These are the common reasons for abnormal liver function tests in pregnancy. Five liver diseases unique to pregnancy includes the following -
    - Hyperemesis gravidarum
    - Intrahepatic cholestasis of pregnancy
    - Preeclampsia
    - Hemolysis, elevated liver enzymes, and low platelet count (HELLP)
    - Acute fatty liver disease of pregnancy
2. **Newly acquired liver diseases** like acute viral hepatitis, drug induced liver injury, or gallstones
3. **Preexisting chronic liver disease** such as cholestatic liver disease, autoimmune hepatitis, Wilson disease, and chronic viral hepatitis.
4. **Physiologic changes in pregnancy** - Abnormal liver function test due to physiological changes in pregnancy without liver dysfunction have a unique pattern.

The most common maternal complications encountered are Encephalopathy, Disseminated intravascular coagulation, Renal failure, Shock, Postpartum hemorrhage, Pyrexia and also Death.

Elevated level of serum bilirubin causes vasoconstrictive effect on the placental vessels and cardiotoxic effect resulting in fetal asphyxia and intrauterine death. Also elevated bilirubin produce cellular effect which stimulates uterine contractility and sensitizes myometrium to oxytocin resulting in preterm labour.

High maternal mortality and morbidity in our country are due to many factors like poor hygiene,inadequate sanitation , malnutrition, prevalence of anemia, delay in seeking medical advice, lack of awareness ,and delay in referral to the higher centers. Many patients are brought in moribund condition to the hospital at admission itself and hence they do not respond to treatment.

The prevalence of viral hepatitis in pregnancy can be reduced by creating public awareness, proper sanitation facilities, safe drinking water, immunization against viral hepatitis, improved antenatal care for early detection and well- equipped hospitals for intensive care. Thereby, mortality and morbidity of jaundice complicating pregnancy can be decreased.

The aim of this study is to identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters and also to determine the feto-maternal outcome among the pregnant women affected by jaundice treated at Government Women and children Hospital, Egmore.

HISTORICAL REVIEW

1. Jaundice is one of the earliest known disease to mankind.
2. The history of jaundice is very long and is described as a sign of causeless hatred in the Babylonian Talmund (Poduri, 2016).

   There are various ancient references related to jaundice which are present in Babylonian Talmund, Sumerian tablets, Ebers papyrus and in ancient Ayurveda (Poduri, 2016)
3. It is documented even in the document of Ebers papyrus dating back to 1500 BC where in first dynasty were found. Ebers papyrus is considered to be the first surviving account of medical remedies.
4. The work of Hippocrates (460 – 370 BC) provides references to jaundice (schmid, 2001, Bynum, 2008)
5. Mc Donald 1908, suggested that jaundice may probably be caused by an agent much smaller than a bacterium (Thomas et. Al, 2015)
6. This idea was developed in 1923 by the hypothesis that virus was the cause for jaundice (Thomas et al, 2013, Poduri 2016).

The words hepatic, liver and jaundice have their origins in Greek, Sanskirt and Old French respectively. Jaundice has its origin in 1300 AD in old French word jaunis that meant “Yellowness”. Subsequently various types of jaundice were described.

OVERVIEW

PHYSIOLOGICAL FUNCTIONS OF LIVER

1. Storage of substances like protein , glycogen , vitamins and folic acid.
2. Synthesis of plasma proteins , glycogen , phospholipids , bile acids and heparin .
3. Secretion of bile acids and bile pigments into the bile.
4. Metabolism of carbhohydrate , protein and fat.
5. Excretion of heavy metals , hormones, cholesterol and bile pigments.
6. Detoxification of ingested drugs.
7. In fetal life the liver produces RBC and WBC.
8. Kupfer cells ACT as immune cells.
9. Thyroxine is converted into triiodothyronin.and also participates in the activation of vitamin D
10. Converts toxic substances into nontoxic substances e.g., benzoic acid is converted into hippuric acid by conjugation with glycine. Ammonia is converted to urea.

PRODUCTION AND METABOLISM OF BILIRUBIN:

SOURCE OF BILIRUBIN:

Mainly 80% is from senescent RBC and about 15 -20 % from ineffective erythropoiesis.

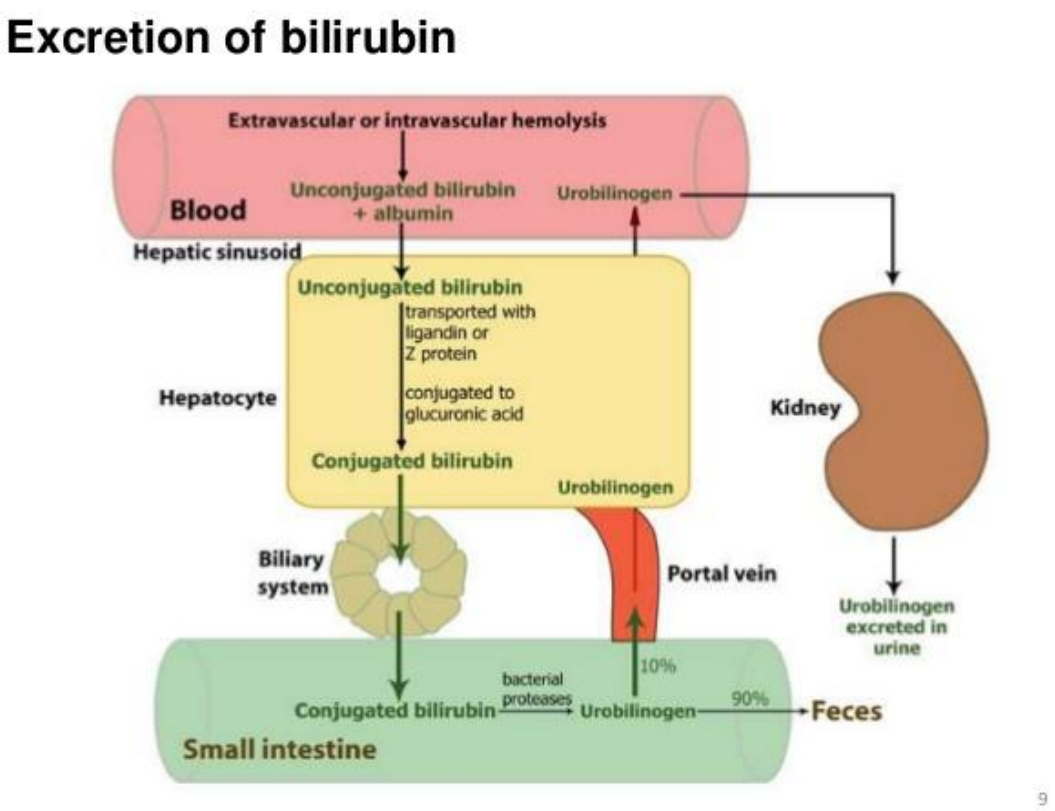
Metabolism of heme containing protein can be divide into three phases.

Hepatic uptake

Conjugation

Excretion into bile (rate limiting step)

1. **BILIRUBIN METABOLISM**



2. PHYSIOLOGICAL CHANGES OF LIVER IN PREGNANCY

a. Liver is not palpable during pregnancy
b. Serum proteins decrease by 20%, Bilirubin is also decreased
c. In third trimester ALP levels increase upto 2 – 4 times the normal. It returns to normal in 2 – 3 months of post delivery
d. ALT and AST level normal, but increase during labour and normalize in 1 – 2 days postpartum.
e. 5” nucleotides is significantly raised but GGT slightly decrease
f. Increase in Sr. Triglyceride and VLDL cholesterol increase upto 2 times the normal.
g. 10 – 15 normal pregnant women may have bilirubin level of over 1% mg due to delayed excretion of bilirubin that may lead to increased incidence of pruritus in pregnancy.

CONCEPTS OF JAUNDICE

Jaundice is a clinical term referring to the yellowish discoloration of Sclera, Mucous Membrane and Skin caused by the accumulation of bilirubin in body fluids. The word jaundice is derived from the old French Word 'Jaunice' meaning yellowness. In medicine `Icterus' derived from Greek is used synonymously (refers to a disease of plants or to a certain yellowish green bird).

Jaundice is clinically detectable when the serum bilirubin concentration exceeds 2.5 mg per dl in the body fluids. Internal tissues are also stained except Brain as bilirubin does not cross the blood - brain barrier. Less marked hyperbilirubinemia is called `Latent Jaundice'.

Mechanisms giving rise to jaundice are:-

1. Increased production - Hemolysis (Lemon Yellow Colour)
2. Impaired excretion
    a. Congenital non hemolytic hyperbilirubinemia
    b. Hepatocellular jaundice (orange yellow colour)
        i. Acute parenchymal liver disease
        ii. Chronic Parenchymal liver diseases 3. Cholestasis (greenish yellow colour)

Other causes of yellowish discoloration of skin (not sclera) are:

1. **Hypercarotenaemia** **2. Mepacrine therapy**

CLASSIFICATION OF JAUNDICE

1. **Based on the Site of Pathology:**

I. PREHEPATIC

Increased production of bilirubin → Hemolytic anaemia

→ Hemolytic reactions

→Tissue infarction

→ Hematomas

→ Post-operative jaundice

→Thalassemia Ineffective erythropoiesis.

II. HEPATIC

1. Impaired Hepatic uptake → Post hepatitis hyperbilirubinemia and storage of bilirubin → Toxic and drug induced hepatitis

→ Portocaval shunts

2. Impaired conjugation of → Gilbert's syndrome
   Bilirubin → Criggler - Najjar syndrome

→Breast milk jaundice

3. Impaired excretion of → Dubin - Johnson syndrome
   Bilirubin →Rotor syndrome

4. Hepato-cellular damage → Recurrent Intrahepatic cholestasis of pregnancy

(Intrahepatic Cholestasis)

→ Viral hepatitis

→ Toxic and drug induced

→ Spirochaetal infections

→ Infectious Mononucleosis

→ Sarcoidosis and lymphomas Hepatitis and hepatic cirrhosis.

III. POST HEPATIC

Biliary epithelial damage → Bile stones

→ Biliary atresia &choledochal cyst.

→ Pancreatitis and carcinoma pancreas.

CLASSIFICATION OF JAUNDICE IN PREGNANCY

(SHERLOCK 1981)

1. Jaundice Peculiar to Pregnancy
   1. Hyperemesis gravidarum
   2. Toxaemias of pregnancy
   3. Recurrent intrahepatic cholestasis of pregnancy
   4. Acute fatty liver of pregnancy.

2. Jaundice not peculiar to pregnancy but superimposed on Pregnancy (Intercurrent Jaundice)
   a. Viral hepatitis (Microbial)
   b. Gall stones
   c. Hepatotoxic drugs
   d. Hemolysis

3. Pregnancy with underlying liver diseases (eg) cirrhosis, familial non hemolytic jaundice

   Microbial Aetiology of Jaundice

   1. Viruses: (90% of microbial causes are due to viruses).
      - Hepatitis virus (HAV, HBV, HCV, HDV, HEV)
      - Arbovirus group B

- Epstein Barr virus
- Cytomegalovirus
- Marburg and Ebola virus
- Rubella
- Enterovirus, coxsackie, echovirus
- Lassa Fever virus
- Herpes simplex virus.

2. Bacterial diseases
    - Leptospirosis
    - Typhoid fever
    - Syphilis
    - Gonococcal disease

3. Parasites
    - Malaria (Plasmodium Falciparum &vivax)
    - Toxoplasmosis
    - Trichomoniasis
    - Amoebiasis

In India the predominance of viral etiology has been quoted by various authors.

The percentage of viral etiology advocated by various authors are

Sanyal and Chowdhary, 1975	-	56%
Issac & Chandrasekar	-	86%
Lahiri, 1976	-	89% and
Devinder Kaur & Ruchi Sharma, 1999	-	66.7%

LIVER FUNCTION TESTS

In a case of jaundice complicating pregnancy, liver function tests should be done

- to confirm the diagnosis
- to assess the severity of the disease and
- to assess the prognosis

The liver functions are depressed in different pathologies in a different way. So all liver function tests should be done ideally. We have to follow certain principles in doing liver function tests.

1. The tests should assess different parameters of liver function.
2. They should be done serially to evaluate the course of the disease.
3. They should be correlated clinically.

TESTS OF BILIARY EXCRETION

1. Serum Bilirubin: Vandenberg's method (1913) of spectrophotometric determination is used.

Normal values are Total: 0.3 - 1 mg / dl

Direct: 0.25 mg% (Conjugated bilirubin)

Indirect: 0.75 mg% (unconjugated bilirubin)

If indirect bilirubin is more than 80% of total bilirubin, it is called predominantly unconjugated hyperbilirubinemia.

If direct bilirubin is more than 50% of the total bilirubin, it is called predominantly conjugated hyperbilirubinemia.

Total values above 2.5 mg/dl is associated with clinical jaundice.

2. Urine Bilirubin (Bile Pigments)

This is estimated by modified Fouchet's test & Hay's Test. Bilirubinuria occurs when there is an increase in the serum conjugated bilirubin. So, it is absent in hemolytic jaundice. Bilirubinuria occurs even with minimal liver damage and can be detected even before clinical manifestation (Isselbacher, 1987).

3. Urine Urobilinogen
    a. It is detected by Ehrlichs Aldehyde test.
    b. It is strongly positive in hemolytic jaundice.
    c. In viral hepatitis, it is positive during pre-icteric and convalescent phase but disappears during icteric phase.

II. TESTS OF METABOLIC FUNCTION

1. **Serum Proteins**:

    Serum proteins are estimated by precipitation of globulin with 23% sodium sulphate. The remaining protein in solution is taken as albumin. The normal serum proteins are Total: 6 - 8.5 gm/dl, Albumin 3.6 - 4.8 g/dl and globulin 2.4 -3.7 g/dl. Normal Albumin - Globulin ratio is 1:5.

    In diffuse parenchymal liver diseases albumin concentration falls and globulin (gamma globulin) fraction increases. The albumin globulin ratio is reversed in such conditions.

2. Serum Alkaline Phosphatase:

    It is estimated by King & Armstrong's method. Normal value is 5 – 15 KA units, it is synthesized by liver, bone intestine, placenta, etc. So, if to be specific, electrophoretic separation of

isoenzymes of alkaline phosphatase is to be done. Marked rise occurs both in intrahepatic and extrahepatic cholestasis.

Clotting Factors

Liver synthesis the clotting factors II, V, VII, IX and X. So, in the presence of severe hepatocellular damage.

1. Clotting time is prolonged (Normal 5 – 15 minutes). The method of Lee and White 1913 is used.
2. Serum prothrombin time: It measures the factors involved in the 2nd and 3rd stages of coagulation in extrinsic coagulation system.

III. TESTS OF LIVER CELL DAMAGE

1. Serum Transaminase:

The liver parenchymal cells contain certain enzymes in large amounts which may be released into the plasma when the cells are damaged. Most important of these is Serum Glutamic pyruvic transaminase (SGPT) or Serum Alanine Aminotransferase (ALT) and Serum Aspartate Aminotransferase (AAT) Normal levels are,

1. SGPT 5 – 35 IU/L
2. SGOT : 5-40 IU/L

With parenchymal liver diseases, the aminotransferase enzymes are elevated sometimes to several hundreds. So serial estimation is needed for follow-up. In patients with acute fulminant hepatitis, serum aminotransferase may decrease due to previous excessive release of these enzymes.

During pregnancy, there are significant normal physiological changes in liver function studies. These include

- A decrease in both the total protein as well as albumin.
- An elevation of the liver dependent clotting factors such as fibrinogen.
- There are elevations in the transport proteins synthesised in the liver such as ceruloplasmin, transferrin and sex steroid binding globulin.
- The alkaline phosphatase will be elevated 2-4 times normal (placenta synthesises Alkaline Phosphatase).
- To note is the fact that transaminase levels should remain normal during pregnancy
- In addition, the bilirubin should stay normal.

JAUNDICE SUPERIMPOSED ON PREGNANCY

VIRAL HEPATITIS

Viral hepatitis is a systemic infection affecting the liver predominantly.

CAUSES OF VIRAL HEPATITIS

• Hepatitis A Virus	Non-A- Non B Non C Viral Hepatitis
• Hepatitis B Virus	Herpes Simplex Virus
• Hepatitis C Virus	Cytomegalovirus
• Hepatitis D Virus	Epstein Barr virus
• Hepatitis E Virus	Yellow Fever virus

Hepatitis B infection is now more frequently seen during pregnancy. This increased incidence may be due to

- Overall increased incidence of the disease.
- May reflect increased sexual abuse and drug abuse and
- More screening for HbsAg.

Normally pregnancy will not affect the course of the disease or make it worse except in 2 situations.

1. In hepatitis E infection and Herpes simplex Hepatitis: Here acute fulminant hepatitis and hepatic failure usually follows.

2. If the liver is already burdened and scarred with cirrhosis, extra demands of pregnancy may predispose to acute fatty liver of pregnancy.

Features of the main Hepatitis Viuses

	Hepatitis A	**Hepatitis B**	**Hepatitis C**	**Hepatitis D**	**Hepatitis E**
Virus Group/ Nucleic Acid/ Size (diameter)	Enterovirus RNA 27nm	Hepadna DNA 42nm	Flavi Virus RNA 30-38nm	Incomplete virus RNA 35nm	Calci virus RNA 27nm
Incubation period (weeks)	2 – 4	4 – 20	2 – 26	6 – 9	3 – 8
Seasonal Incidence	Winter	Year around	Year around	Year around	Winter
Age	Children	Any	Any	Any	Any
Spread 1. Feco-oral 2.Blood 3.Sexual 4.Vertical	 Yes Uncommon Uncommon No	 No Yes Yes Yes	 No Yes Uncommon Uncommon	 No Yes Yes Yes	 Yes No ? No
Type of onset	Acute	Insidious	Insidious	Acute or Insidious	Common
Carrier State	No	Yes (5-10%)	Yes >50%	Yes	No
Prognosis	Good	Worse with age	Moderate	Same as HBV	Good except in pregnancy
Prevention Active Passive	Vaccine Immune Serum Globin	Vaccine Hyperimmune Serum Globin	No No	Prevented by Prevention of Hepatitis B Virus Infection	No No

Clinical Features

1. Prodromal Phase
    - Precedes jaundice by few days to 2 weeks
    - Symptoms include fever, malaise, arthralgia, headache, nausea, vomiting, diarrhoea, anorexia, and upper abdominal pain.
    - Hepatic tenderness present but spleen & liver not palpable. Urine SGOT and SGPT shows variable increase; serum bilirubin is usually normal.

2. Icteric phase
    - Prodromal symptoms diminish and jaundice is visible with dark urine and yellow sclera; clay stools may occur.
    - Liver is palpable and tender.
    - Splenomegaly and cervical lymphadenopathy is present in 1-20% of cases.
    - Transient spider angiomas may appear.
    - Serum bilirubin is elevated. SCOT and SGPT markedly elevated. Serum Alkaline phosphatase is also increased. Some may show hypoglycemia. Leukopenia with lymphocytosis can occur.

3. Convalescent phase

    - Constitutional symptoms disappear and appetite improves.
    - Urine and stool become normal; sclera takes a long time to become normal.
    - Liver regresses slowly.
    - Biochemical recovery is late; SGOT and SGPT decreases but serum bilirubin may rise. • Complete recovery is within 2-3 months in uncomplicated cases.

ANTIGENIC RESPONSE TO VIRAL INFECTION

Hepatitis A infection

- Hepatitis A virus (HAV) antigen is found & is present transiently in the blood during incubation.

Hepatitis B infection

Three antigens are important - surface Antigen (HBsAg), core antigen (HBcAg) and e antigen (HBeAg).

- The HBsAg is the first to appear - late in the incubation period, disappears usually in one month but may persist for 3-5 months.
- HBcAg is not demonstrable in the serum.
- HBeAg appears in the early illness and it is a marker of infectivity.

- Antibodies form in a week or two after the onset of jaundice - the order is first core antibody, then e-antibody and much later surface antibody.

Interpretation of Serological Tests in Hepatitis B virus infection

Interpretation	**HBsAg**	**Anti HBc**		**Anti HBS**
		IgM	**IgG**	
Incubation period	+	+	-	-
Acute Hepatitis				
Early	+	+	+	-
Established (occasional)	+	+	+	-
Convalescence (3-6 months) (6-9 months)	 - -	 ± -	 + +	 ± +
Post Infection >1 Year Uncertain	 - -	 - -	 + +	 + -
Chronic Infection Usual Occasional	 - -	 - -	 + +	 - -
Immunisation without infection	-	-	-	+

Hepatitis C virus infection:

Many antigens and antibodies are available but are unreliable.

Hepatitis B virus infection:

A single antigen which is transient but anti HDV can be detected in the blood.

Hepatitis E virus:

Anti HEV is used for diagnosis.

Certain observations during various studies are

- There is an increase in the incidence of hepatitis with advancing stages of pregnancy
- The hepatitis infection predisposes the woman to preterm delivery.
- Poor prognosis has been noted if the onset of hepatitis is in the third trimester. The high mortality rate with hepatitis in pregnancy was first pointed out by Zondek and Bromberg. The role of hormonal and metabolic changes of pregnancy as contributing factors was stressed by Dill et al. However, malnutrition definitely worsens the prognosis.
- Low prothrombin level in hepatitis may predispose the woman to post-partum haemorrhage.

- Fetal wastage in viral hepatitis: various studies have reported the increased incidence of abortion, preterm deliveries, still birth and neonatal deaths, more in fulminant cases.

Hepatitis A and pregnancy

- It is typically self-limiting and does not appear to carry a different prognosis than that in the nonpregnant.
- The teratogenic effects of acute HAV during pregnancy have not been noted.
- Vertical transmission however is rarely reported.
- But transmission to the neonates from an infected mother can occur by the usual feco-oral route during delivery and the post-partum period.
- If the new born is exposed, the infection is usually mild and they will have a lifelong immunity to the disease.
- Even then new born infants of HAV infected mothers whose symptoms first manifested between 2 weeks before and 1 week after delivery should receive supplemental immune globulin.
- If the pregnant woman is exposed, she can be given immunoglobulin to protect her. The safety of Hepatitis A vaccine is not proved during pregnancy.

Hepatitis B and pregnancy

Various studies show that the clinical course and histologic findings of chronic HBV infection do not differ between pregnant and non-pregnant patients.

Perinatal transmission occurs primarily as a result of oral contamination of baby with infected blood and genital secretions during delivery. If the mother is positive for both HBsAg and HBeAg, the risk of transmission is 70-90% and 85% or more of children ultimately become carriers of HBsAg.

For carriers of HBsAg alone, the rate of transmission is 10-20%. This suggests that active HBV particles are unlikely to cross the intact placenta. Placental breaks in the maternal fetal circulation are of course possible, particularly at the end of pregnancy.

Factors that increase the risk of vertical transmission include

- ➔ Concurrent HIV infection
- ➔ High maternal viral load
- ➔ High titres of HBsAg
- ➔ Presence of HBeAg.

The neonatal infection is rare if maternal infection takes place in the first trimester. The neonatal infection rate is 6% in the second trimester and 67% in the third trimester and more in the immediate post-partum period. It has been appreciated that the finding of a mother who is a carrier for hepatitis B places her foetus at risk in later life of developing both chronic hepatitis and hepatocellular carcinoma.

Infants of seropositive mothers should receive.

- Hepatitis B Immunoglobin 0.5 ml IM immediately after birth.
- Hepatitis B vaccines should be given at three shots
    - 1st dose within 12 hours after birth
    - 2nd dose at 2 to 4 months
    - 3rd dose at 6 to 18 months.

Neonatal immuno-prophylaxis is 85 to 95 percent effective in preventing neonatal hepatitis B infection.

Hepatitis C infection and pregnancy

Pregnancy does not appear to induce deterioration of liver disease in women with HCV, nor HCV increases the risk of obstetric complications.

Vertical transmission of HCV

1. If the woman is HIV positive, 5% risk.
2. If the woman is HIV negative or RNA negative, risk is unlikely
3. If the woman is HIV positive or high level of RNA positivity (> 1 million copies/1W) 35% risk.

At present no Hepatitis C vaccine is available. The suspected infant should undergo HCV RNA screening at 12 months and if positive is to be treated with interferon. Little is known about Hepatitis D infection.

HERPEX SIMPLEX HEPATITIS IN PREGNANCY

- Predominant in pregnant women.
- Virus is Herpes simplex type 2.
- Systemic symptoms are common.
- Characteristic mucocutaneous lesions appear.
- Serum bilirubin is mildly elevated but serum transaminase is markedly elevated.
- Maternal and fetal mortality are about 50%
- Acyclovir is the treatment of choice.

COMPLICATIONS OF HEPATITIS:

- Fulminant hepatic failure, coma and death
- Relapsing hepatitis (Biochemical & clinical)
- Cholestatic hepatitis
- Post hepatitis syndrome
- Hyperbilirubinemia
- Aplastic anaemia
- Connective tissue disease
- Renal failure
- Chronic hepatitis, cirrhosis, hepatocellular carcinoma.
- Disseminated intravascular coagulation.

Management

1. Bed rest improves hepatic circulation especially if symptoms are marked.
2. Nutrition: Nutritious diet containing 3000 kcal/day is recommended. A light diet with fruit drinks and glucose is advisable. If vomiting is severe, intravenous glucose is to be given. Vitamins can be given.

3. Drugs

    - Interferons are not recommended
    - Alcohol should be stopped during the illness and the following 6 months.
    - Drugs metabolised in the liver (sedatives and hypnotics to be avoided).
    - Oral contraceptives may be resumed after clinical and biochemical recovery.
    - Constipation, diarrhoea, hypokalaemia, infections and diuretics are to be avoided.
    - Supportive measures if the patient develops hepatic coma along with Vitamin K and fresh blood transfusion.

Cholelithiasis in pregnancy

It is noted in 6% of pregnant women. Pregnancy predisposes to gallstones because

- Bile salt pool decreases in the second trimester.
- Biliary cholesterol levels may increase.
- Gall bladder emptying slows in the second trimester.

Suggestive clinical features

- Pain abdomen, fever, nausea & vomiting.
- Tenderness in the Murphy's point (tip of the 9th costal cartilage on the right side).
- Elevated serum bilirubin and serum transaminase levels but Serum Alkaline Phosphatase level is unreliable.
- Ultrasonogram is diagnostic showing Gallbladder sludge or stone.

Treatment

Laparoscopic cholecystectomy can be safely done during the first and second trimesters but to be avoided in the third trimester.

PREGNANCY SPECIFIC CAUSES

1. **Intrahepatic cholestasis of pregnancy**

- It is caused by an idiosyncratic exaggeration of normal hormonal effects of bile transport. There is a combination of defects in the sulfation of oestrogens and canalicular excretion of these metabolites.
- There is racial and familial predisposition.
- Typically starts in the third trimester (20-35 weeks)
- Pruritus and jaundice are seen.

- Elevated serum bilirubin levels, mild elevation of aminotransferase, marked elevations of bile acids are present.
- Liver biopsy shows centrilobular bile stasis without inflammatory cell infiltrate.
- Symptoms resolve within 2 days after delivery: Biochemical normality is at 4-6 weeks after delivery.
- Cholestasis recurs in 60-70% of subsequent pregnancies. Prolonged pruritis has been described and also one case of cirrhosis.
- Fetal wastage: Preterm delivery 20%, Meconium staining & Fetal distress 25%
- Management
    1. Urso-deoxycholate decreases pruritus (20-25 mg/kg/day), ameliorates liver enzymes (?), improved fetal outcome
    2. Dexamethasone Same effect
    3. Phenobarbital & cholestyramine are not helpful.
    4. Frequent fetal monitoring
    5. Delivery - at 38 weeks; at 36 weeks if jaundiced.

Hyperemesis gravidarum

Pernicious nausea and vomiting may produce hyperbilirubinemia, mild to moderate liver enzyme elevation. Sheila Sherlock has stated that as a result of fat mobilization from depot due to malnutrition, fat deposits in the liver and produces slight fatty metamorphosis; jaundice is unusual. After relief of hyperemesis or after delivery liver becomes normal.

ACUTE FATTY LIVER OF PREGNANCY

- It is a life - threatening condition with an 18% MMR and 23% perinatal mortality rate. Incidence is rare (1 in 13,000), common in nullipara.
- Most frequently complicates the third trimester.
- Symptoms include anorexia, nausea, vomiting and progressive jaundice.
- Laboratory findings include elevated serum bilirubin levels of 1 to 10 mg%, Serum transaminase levels of 300 - 500 IU/L, hypofibrinogenemia, marked hypoglycemia, peripheral smear showing Echinocytes and evidence of hemolysis.
- Hepatic histopathology reveals pericentral micro vesicular fat with minimal inflammation or necrosis. Liver biopsy is not indicated for diagnosis. Usually renal changes are associated.

- Acquired abnormalities of mitochondria or intermediary metabolism of fatty acids or both may be the cause. Recently familial deficiency of long chain 3 hydroxy acyl - COA dehydrogenase (LCHAD) has been described. In this condition only, it may recur in subsequent pregnancies.
- Maternal complications include Pre-eclampsia, hypofibrinogenemia and coagulopathy (55%), Renal Failure, severe hepatic dysfunction, hypovolemia and acidosis, hepatic coma, acute pancreatitis, gastrointestinal bleeding and death. (Diabetes insipidus has been reported).
- Increased rate of intrauterine death is noted. Some children express homozygous deficiency of the enzyme, producing severe metabolic and neurologic consequences.
- Treatment: Expeditious delivery, intensive care & FFP transfusion.
- Patients usually improve promptly following delivery and in the absence of LCHAD, the prognosis in pregnancies is better. No subsequent recurrence.

PRE ECLAMPSIA

- Recently, hepatic dysfunction has been associated with other findings in the HELLP syndrome. It complicates 3-10 % of patients with pre eclampsia and 0.1 % of all pregnancies. Common in multipara. MMR is 2%. PNMR is 33%.
- Pathophysiology: vasospasm, endothelial damage and Platelet dysfunction. Recently defect in nitric oxide metabolism has been attributed.
- Characteristic abnormalities include:
    1. Hemolysis – eleveted bilirubin levels and lactate dehydrogenase levels greater than 600 IU/L, peripheral smear showing Schistocytes, fragmented RBC s.
    2. Moderately elevated transaminase levels (AST and ALT levels of 200 – 700 IU / L).
    3. Platelet count less than 1,00,000 per ml.
- Typical presentation is right upper quadrant pain and malaise; 60% have edema; 50% have nausea or vomiting.
- No correlation between extent of hypertension, liver function test abnormalities or liver biopsy findings.

- Notable complications are coagulopathy, hepatic failure, multiple organ failure, rarely rupture of a liver hematoma.
- Prompt delivery is the effective treatment. Postpartum corticosteroids improve maternal platelet count and ALT levels. Following delivery, lab abnormalities peak in the first one to two days and return to normal in 1 to 2 weeks.
- Risk of recurrence is 3.4%.

LEPTOSPIROSIS (WEILS DISEASE)

- It is an infectious disease caused by the spirochete - Leptospira icterohaemorrhagiae. Rat is the natural host. Infected urine contains spirochetes which penetrate the skin or mucosa of humans.
- Disease begins abruptly with pyrexia, conjunctival suffusion, headache, and petechial rashes.
- Hepatitis, renal tubular necrosis, myocarditis and meningitis may complicate.
- Severe cases may progress to liver failure, renal failure, cardiac failure and death (15-20%).
- If recovery occurs it is complete (Third and Fourth week).

- Rising titre of 'specific leptospiral antibodies from the second week is diagnostic.
- Management: Benzyl penicillin 1.2 g IV or IM 6th hourly for 10 days is the treatment of choice. Alternative is erythromycin 500 mg 12 hourly.

PREGNANCY AND CHRONIC LIVER DISEASE WITH PORTAL HYPERTENSION

Portal hypertension occurs due to the obstruction of flow within the portal circulation which may be presinusoidal, hepatic sinusoidal or post sinusoidal.

In cases with presinusoidal PHT and pre-cirrhotic parenchymal liver disease, architecture and liver function is preserved and they are fertile. In advanced chronic liver disease, there is decreased fertility, decreased libido and there is physical distress.

Effect of Portal Hypertension on the Pregnancy outcome

Spontaneous abortion (15-20%) is common.

Causes of Termination before 20 weeks

1. Spontaneous abortion
2. Therapeutic abortion

Causes of termination after 20 weeks

1. Variceal haemorrhage in the mother
2. Therapeutic abortion.

Effect of pregnancy on portal hypertension

1. Symptomatic anaemia occurs in 7% to 9%
2. Maternal mortality is increased 5% to 7% in extrahepatic PHT

   10% to 18% in cirrhosis

Causes of maternal mortality are

1. Variceal haemorrhage (II trimester and during labour).
2. Liver failure.
3. Hepatic encephalopathy.
4. Post-partum haemorrhage (7 to 10%) due to coagulopathy or hypersplenism and thrombocytopenia.
5. Spontaneous bacterial peritonitis
6. Rupture of splenic artery aneurysm
7. Rupture of splenorenal shunts.

Management

Needs coordinated approach by the hepatologist and obstetrician.

Prenatal Management before conception

1. Risk assessment for pregnancy.
2. Contraceptive advice : Oral contraceptive pills or long acting medroxy progesterone.
3. Management of portal hypertension:
    - No prior history of bleeding
        i. non selective Beta blockers (Risk : fetal bradycardia and IUGR).
        ii. Isosorbide mononitrate
    - Previous history of bleeding
        i. Portal decompression or ii. Endoscopic therapy
4. Counselling regarding vertical transmission of infectious liver diseases.
5. Evaluation of risks of the teratogenic potential of medications.

Antenatal Management after conception

1. **Management of Pregnancy**

    a. Hemogram, renal and liver function tests

    b. Iron and multivitamin supplementation

    c. Fetal monitoring

    d. Immunisation against HBV

2. **Management of Portal hypertension**

    Beta blockers or endoscopic treatment or portal decompression. Sodium restriction, management of cause of portal hypertension. Ultrasound examination for splenic Artery aneurysm to be done.

Management of Labour

1. Analgesia in the I stage (to avoid premature bearing)
2. Over sedation is to be avoided
3. Shorten II stage of labour with outlet forceps
4. Avoidance of volume overload
5. Caesarean section for emergency situations (Abdominal and Pelvic collaterals may bleed)

Prognosis is bad with alcoholic cirrhosis. In Biliary cirrhosis, increased cholestasis may aggravate itching. Other immune mediated cirrhosis improves with pregnancy.

REVIEW OF LITERATURE

Outcomes of pregnancies complicated by hyperemesis gravidarum.

A population-based retrospective cohort study was conducted by Dodds et al among women with singleton deliveries between 1988 and 2002. Maternal outcomes evaluated included weight gain during pregnancy, gestational diabetes, gestational hypertension, labor induction, and cesarean delivery. Neonatal outcomes included 5-minute Apgar score of less than 7, low birth weight, small for gestational age, preterm delivery, and perinatal death.The study concluded that the adverse infant outcomes associated with hyperemesis are a consequence of, and mostly limited to, women with poor maternal weight gain.

Association of adverse perinatal outcomes of intrahepatic cholestasis of pregnancy with biochemical markers: results of aggregate and individual patient data meta-analyses

Caroline Ovadia et al observed in a meta-analysis study that the risk of stillbirth is similar in women with intrahepatic cholestasis of pregnancy and singleton pregnancies when serum bile acids concentrations are less than 100 μmol/L. The risk is increased in women with intrahepatic cholestasis of pregnancy and singleton pregnancies when serum bile acids concentrations are of 100 μmol/L or more.

Predictors of adverse neonatal outcomes in intrahepatic cholestasis of pregnancy.

A multicenter retrospective cohort study of all women diagnosed with ICP conducted by Kawakita et al concluded that women with ICP with serum

bilirubin level ≥100 μmol/L had an increased risk of stillbirth. Whereas a bilirubin level ≥40 μmol/L was associated with increased risk of meconium-stained amniotic fluid.

A comparative study of maternal and perinatal outcome in patients with and without HELLP syndrome

A prospective study of feto maternal outcome in severe preeclampsia and eclampsia with and without HELLP syndrome. 83.7% of cases among HELLP group had normal maternal outcome. 11.6% maternal mortality among the HELLP syndrome group. 54.5% babies among HELLP group had abnormal perinatal outcome and 24.6% among non HELLP group.

Maternal and fetal outcome of pregnancy complicated by HELLP syndrome.

Gasem et al evaluated the maternal and fetal outcome in 64 pregnancies complicated by HELLP syndrome.42% of the patients had deliveries <32 weeks and 28% IUGR. Respiratory distress syndrome was the main indication for NICU admissions (33.9%). The PNM rate was 20%. Maternal morbidity rate was 34%. The most common maternal complications were abruptio placentae (36.4%) and DIC (31.8%).

Maternal pre-pregnancy infection with hepatitis B virus and the risk of preterm birth: a population-based cohort study

Jue Liu et al conducted a population based cohort study on 489965 women in China.The study yielded the following results: Compared with women who were not infected with hepatitis B virus, women who were HBsAg

positive and HBeAg negative had a 26% higher risk of preterm birth (aRR 1·26, 95% CI 1·18–1·34) and women who were both HBsAg and HBeAg positive had a 20% higher risk of preterm birth (aRR 1·20, 1·08–1·32). Comared with women who were not infected with hepatitis B virus, women who were HBsAg positive and HBeAg negative manifested an 18% higher risk of early preterm birth (gestation less than 34 weeks; aRR 1·18, 1·04–1·34) and women who were both HBsAg and HBeAg positive had a 34% higher risk of early preterm birth (aRR 1·34, 1·10–1·61). Maternal pre-pregnancy hepatitis B virus infection was independently associated with higher risk of preterm birth and early preterm birth.

Fetomaternal outcome in acute hepatitis e.

Sultana R and Humayun S conducted a study between the period July2012-March 2013.The mean gestational age was 32 weeks. Twenty one (84%) babies were born alive, among them 18 (86%) were preterm. Perinatal mortality was 26%; contributed by intrauterine deaths and early neonatal deaths in 3 (14%) cases each. Total maternal deaths were 5 (20%), 4 (80%) in postpartum period and 1 (20%) in antepartum period due to fulminant hepatic failure in all cases. According to this study prematurity in newborns and fulminant hepatic failure in mothers are major cause of poor fetomaternal outcome in acute hepatitis E in pregnancy.

A Study of Hepatitis E in Pregnancy: Maternal and Fetal Outcome

Gowri Sayi Prasad et al studied the pregnancy outcomes of Anti-HEV IgM-positive women. The maternal mortality was 5 % including one antenatal

death. Prematurity (80 %) and PROM (11 %) were the commonest fetal complications noted with a vertical transmission rate of 28 %.

Outcomes of gallstone disease during pregnancy: a population based data linkage study

Ibinabo Ibiebele et al published the following results:

Gallstone disease was associated with increased risk of preterm birth (aRR 1.3, 99% CI 1.1, 1.6) particularly planned preterm birth (aRR 1.6, 99% CI 1.2, 2.1), morbidity (aRR 1.6, 99% CI 1.1, 2.3), maternal readmission (aRR 4.7, 99% CI 4.2, 5.3), and neonatal morbidity (aRR 1.4, 99% CI 1.1, 1.7). Surgery was associated with decreased risk of maternal readmission (aRR 0.4, 99% CI 0.2, 0.7).

Acute hepatitis A infection in pregnancy is associated with high rates of gestational complications and preterm labor.

Elinav E et al published the following observations in this study.

Acute HAV infection during pregnancy is associated with high risk of maternal complications(premature contractions, premature separation of membranes and vaginal bleeding) and preterm labor.

Clinical characteristics and gestational complications associated with acute hepatitis a in pregnancy.

A retrospective study by Ryu HS et al inferred that acute HAV infection during pregnancy may be associated with the risk of gestational complications.

AIMS AND OBJECTIVES

1. To analyze the maternal outcome in terms of mode of termination of pregnancy, maternal morbidity and mortality in jaundice complicating pregnancy,

2. To identify the relation of maternal morbidity and mortality in relation to admission serum bilirubin level.

3. To assess fetal outcome by perinatal mortality and morbidity.

4. To identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters

INCLUSION CRITERIA:

All pregnant women affected by jaundice treated in Government Women and Children Hospital, IOG, Egmore.

EXCLUSION CRITERIA :

Jaundice in pregnant women occuring due to septic etiology.

STUDY CENTRE: Institute of Obstetrics and Gynaecology, Chennai

MATERIALS AND METHODS:

79 patients admitted with c/o jaundice in all trimesters of pregnancy admitted to the Government Hospital For Women and Children, IOG, Egmore during the period of January 2018 to February 2019 were studied in detail.

A detailed history including patients age, socio economic status, booking, parity and details of menstrual history, previous obstetric history, last menstrual period were obtained to arrive at the expected date of delivery.

Past history including history of Jaundice in the previous pregnancy, history of contact with jaundice, history of injection, blood transfusion, history of intravenous drug abuse, history of any hepatotoxic drug intake and family history of Jaundice were obtained.

In the present history, symptoms like fever, Nausea, vomiting, malaise, loss of appetite, diarrhoea, arthralgia, colour of urine and motion, pruritus, history of bleeding episodes were asked for. The above data were collected in a structured proforma.

Thorough general and systemic examination was carried out for each patient. In addition, evidence for anaemia, hypertension, Hepatomegaly, splenomegaly and purpuric spots were carefully recorded.

A detailed systemic examination was done.

Investigations included urine albumin, sugar, deposits, urine for culture sensitivity, bile salts and bile pigments, motion for ova and cyst, hemoglobin, RBC count, PCV, Total count, Differential count, Platelet count, peripheral smear for parasites, Immature and abnormal cells, type of anaemia, bleeding time, clotting time, blood VDRL, Blood WIDAL, liver function tests including Vandenberg reaction, serum enzyme levels of aspartame transaminase, alanine transaminase and alkaline phosphatase, serum total proteins, albumin and globulin ratio, serum fibrinogen, blood urea and sugar, serum creatinine, blood grouping and typing.

Examination for leptospirosis was done when fever was associated with purpuric spots.

On admission, 15 ml of blood was drawn by venipuncture with all aseptic precautions. 5 ml was sent for liver function test. 2 ml for complete hemogram,1ml for blood grouping and 5 ml for vital serological marker study.

Liver function test was repeated every week in all the patients till the time of recovery and discharge from the hospital. The patients were followed up for pregnancy outcome.

The diagnosis of viral Hepatitis was done by clinical suspicion and presentation. The only serological marker done was HbsAg. Likewise the diagnosis of intra hepatic cholestasis of pregnancy and acute fatty liver were done by clinical presentation and biochemical parameters.

ANALYSIS AND RESULTS

TABLE 1: SYMPTOMS

Symptoms	Frequency	Percent
High colored Urine	51	44.3
History of fever	47	40.9
Loss of appetite	43	37.4
Nausea & Vomiting	47	40.9
Upper abdominal pain	14	12.2
Itching	4	3.5
Loose stools	2	1.7
Clay stool	1	0.9

CHART 1: SYMPTOMS

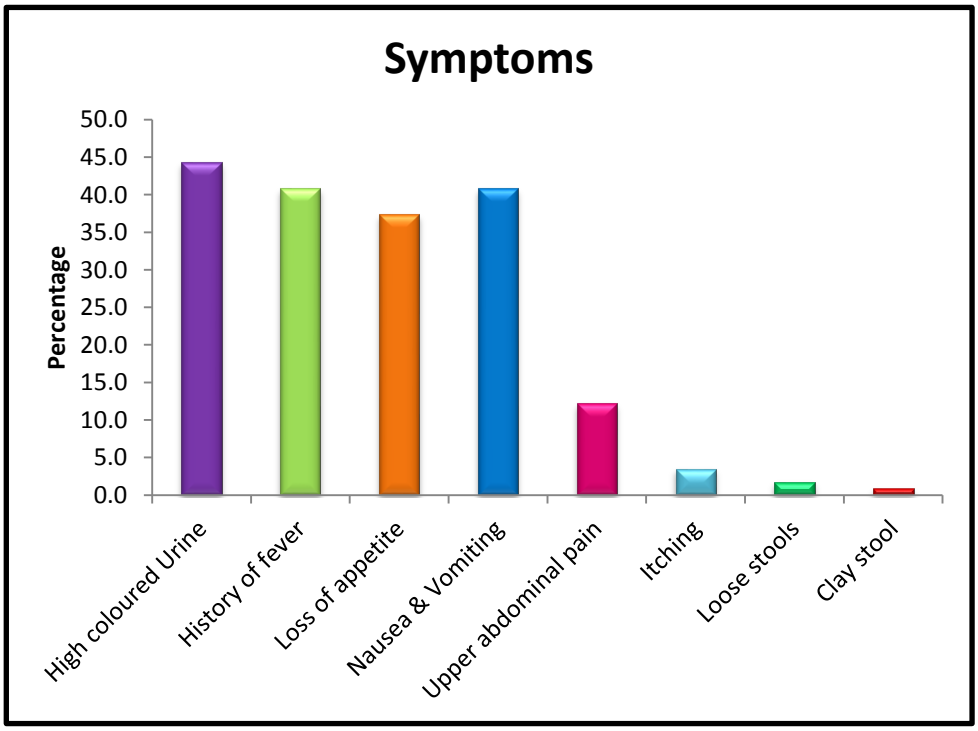


TABLE 2: SIGNS

Signs	Frequency	Percent
Jaundice	70	60.9
Hepatomegaly	19	16.5
Splenomegaly	7	6.1
Scratch marks	4	3.5
Ascites	1	0.9

CHART 2: SIGNS

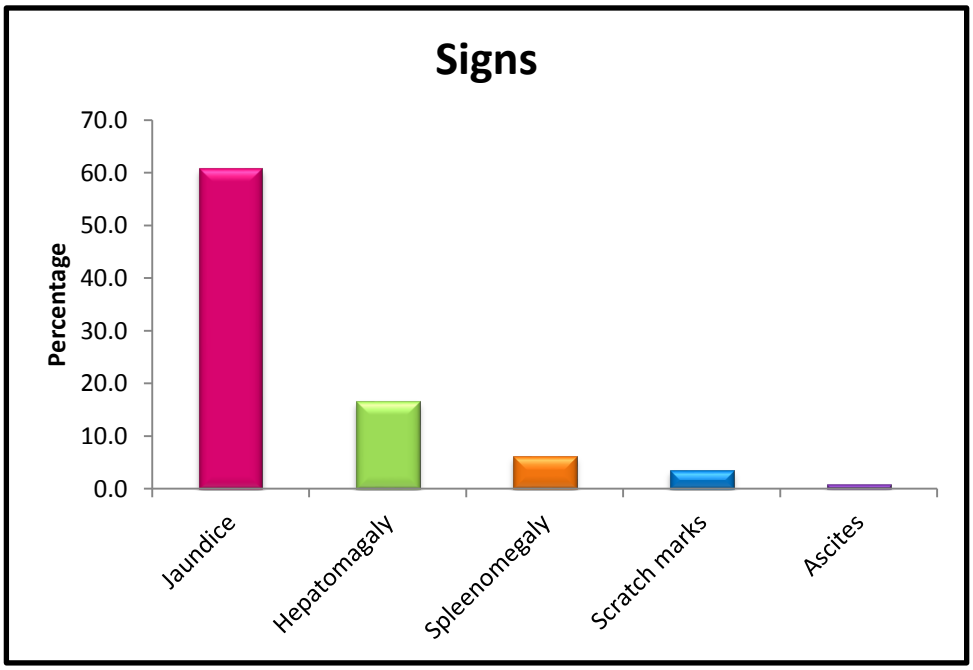


CHART 3: COMPLICATIONS

Complications	Frequency	Percent
Anemia	20	25.4
Pre-eclampsia	6	7.6
Hematemesis	5	3.8
Encephalopathy	3	3.8
Hepatorenal failure	4	5.1
Nil	47	59.5

CHART 3: COMPLICATIONS

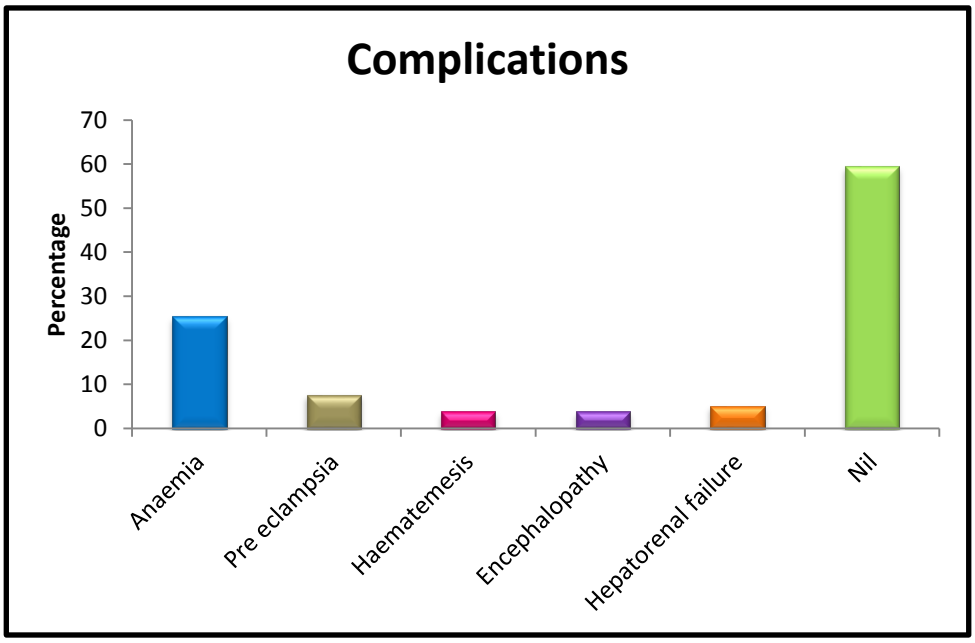


TABLE 4: AGE

Age	Frequency	Percent
18 - 20 yrs	12	15.2
21 - 25 yrs	44	55.7
26 - 30 yrs	18	22.8
31 - 35 yrs	3	3.8
> 35 yrs	2	2.5
Total	79	100.0

CHART 4 : AGE

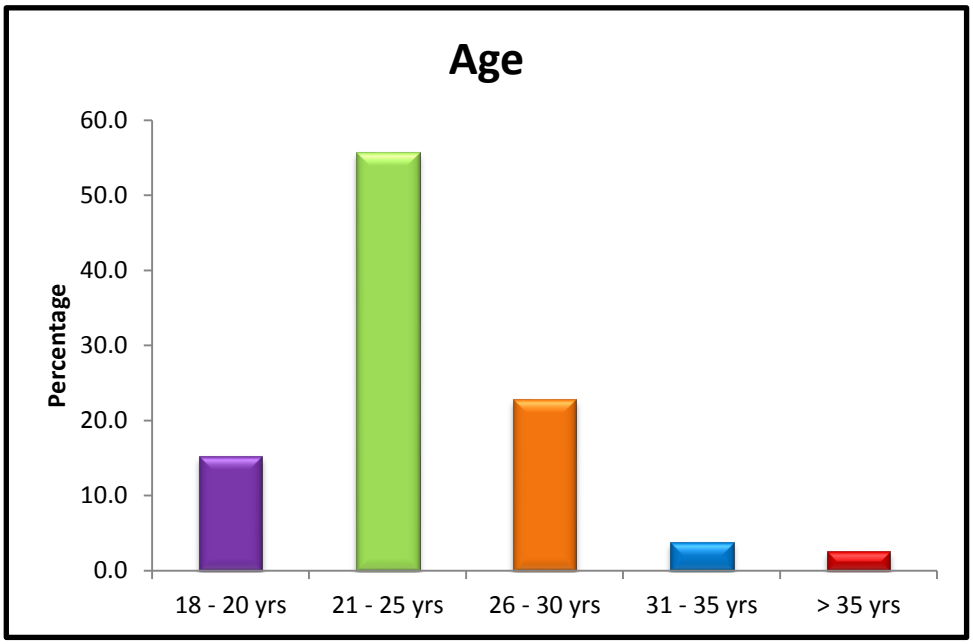


TABLE 5: OBSTETRIC HISTORY

Obstetric History	Frequency	Percent
Multi	45	57.0
Primi	34	43.0
Total	79	100.0

CHART 5: OBSTETRIC HISTORY

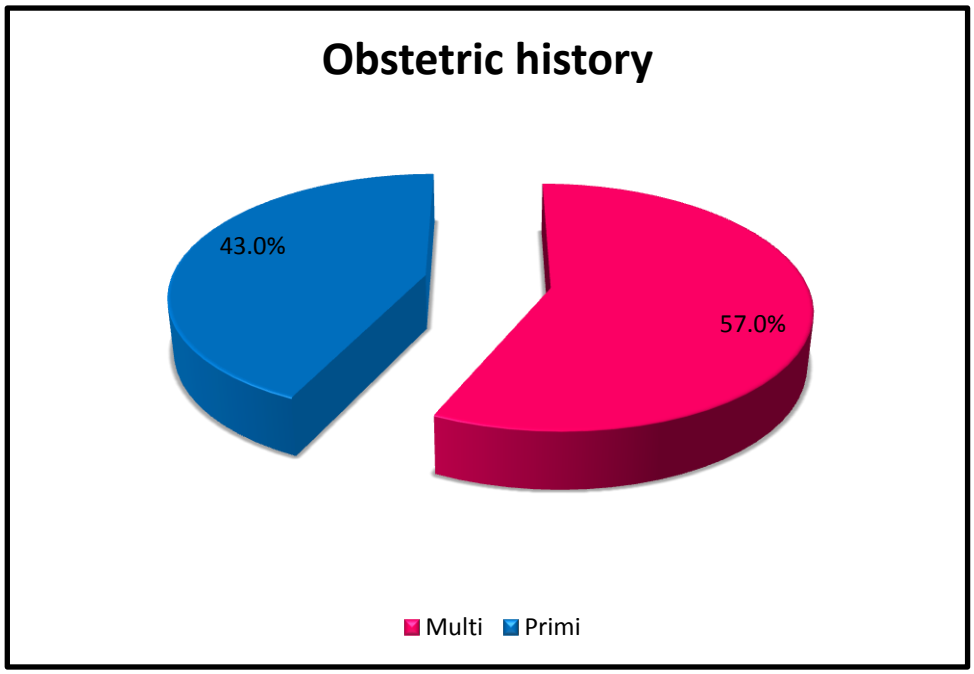


TABLE 6: DURATION

Duration	Frequency	Percent
Upto 5 days	28	35.4
6 - 10 yrs	42	53.2
11 - 30 days	5	6.3
> 30 days	4	5.1
Total	79	100.0

CHART 6: DURATION

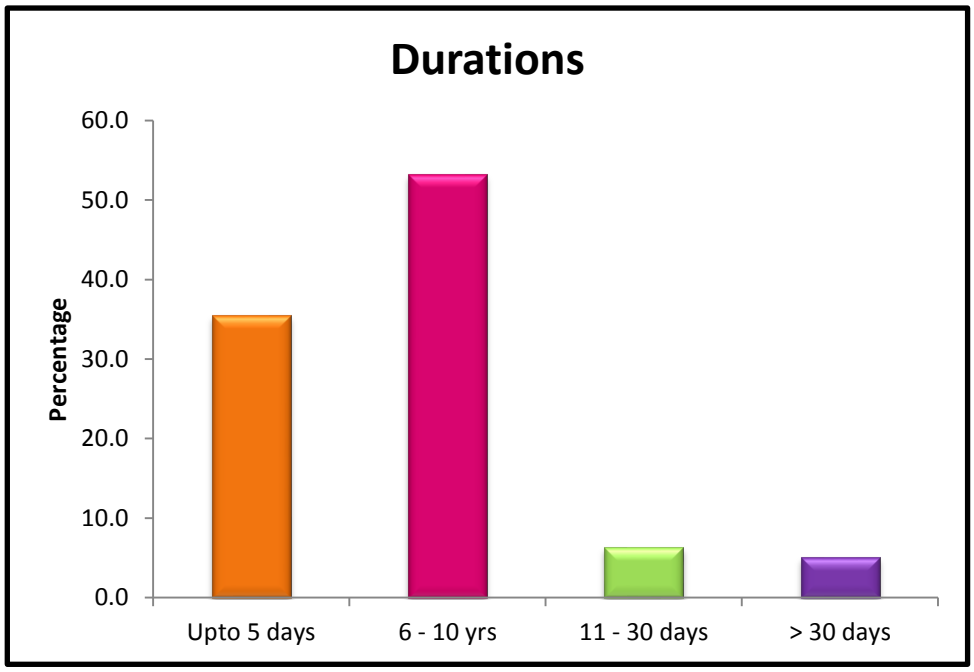


TABLE 7: BS

BS	Frequency	Percent
Negative	4	5.1
Positive	75	94.9
Total	79	100.0

CHART 7: BS

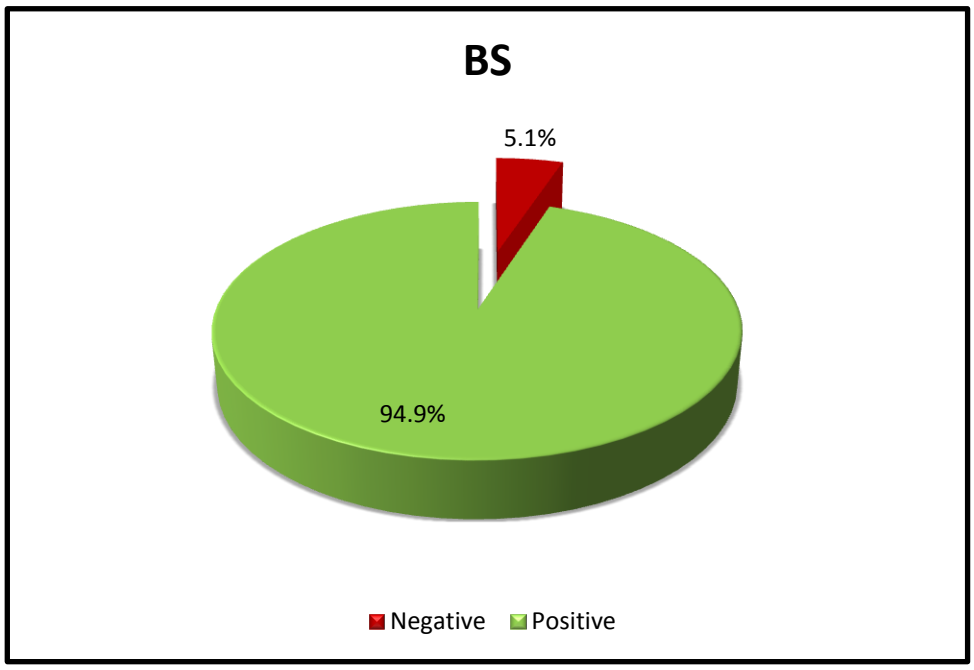


TABLE 8: BP

BP	Frequency	Percent
Negative	4	5.1
Positive	75	94.9
Total	79	100.0

CHART 8: BP

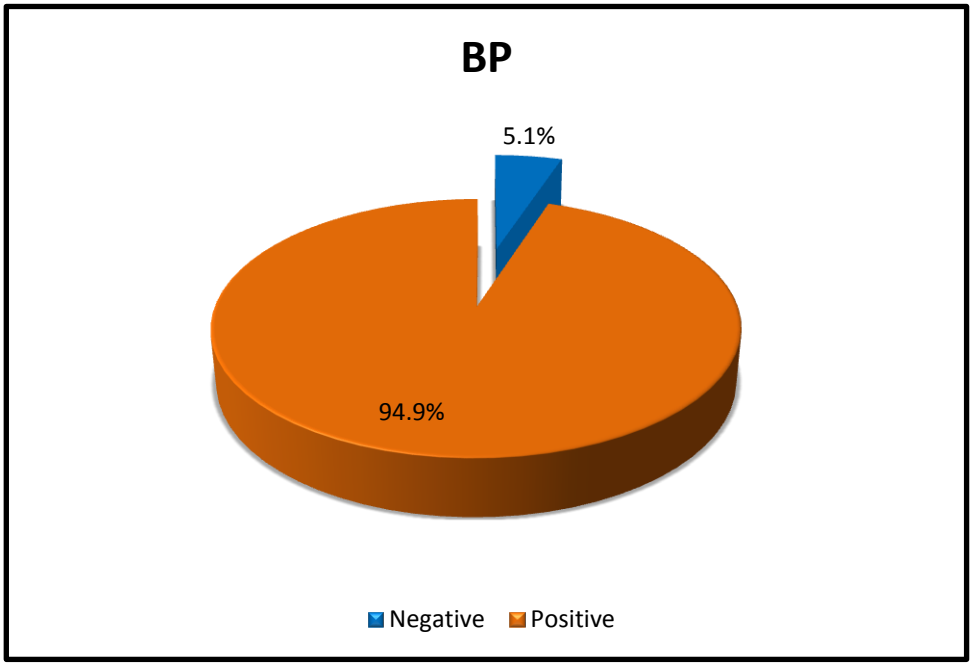


TABLE 9: PROTEIN

Protein	Frequency	Percent
Negative	72	91.1
Positive	7	8.9
Total	79	100.0

CHART 9: PROTEIN

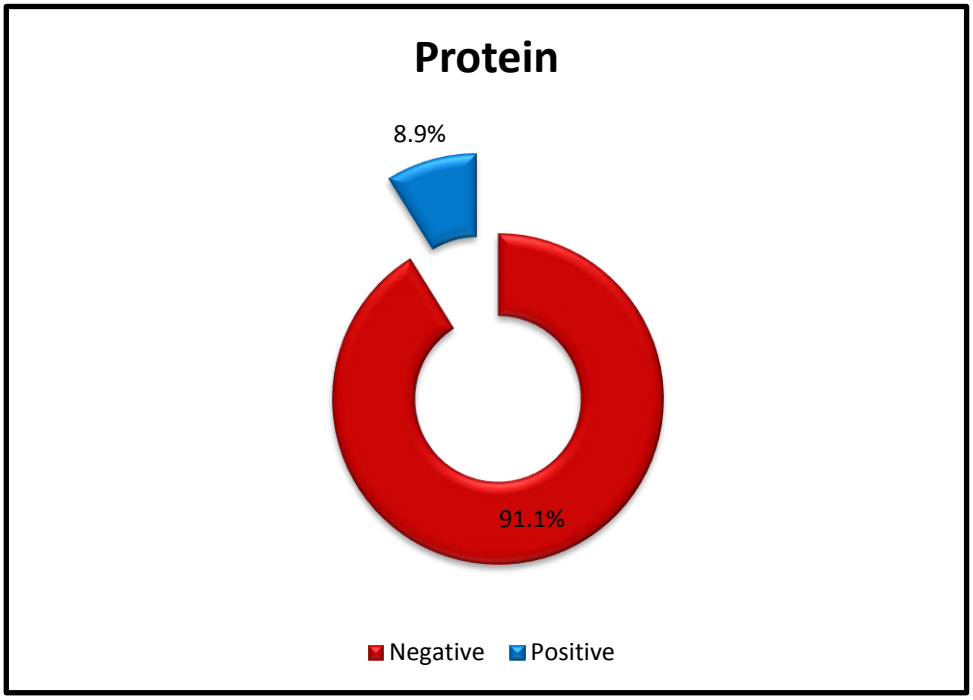


TABLE 10: SUGAR

Sugar	Frequency	Percent
Negative	79	100.0

CHART 10: SUGAR

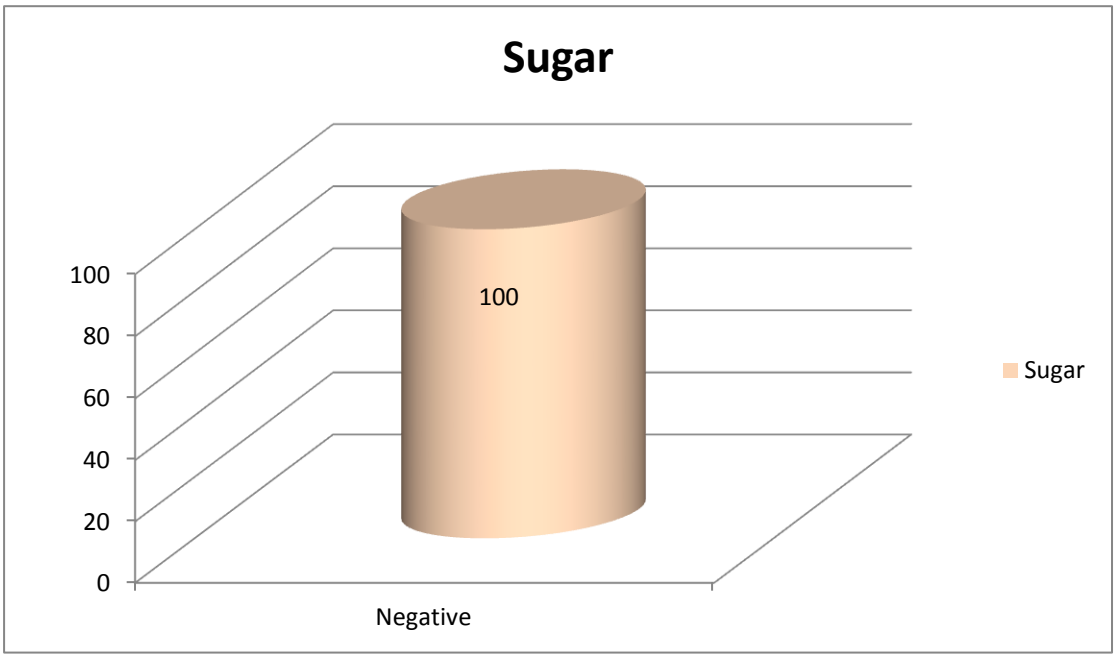


TABLE 11: HbsAg

HbsAg	Frequency	Percent
Negative	62	78.5
Positive	17	21.5
Total	79	100.0

CHART 11 : HbsAg

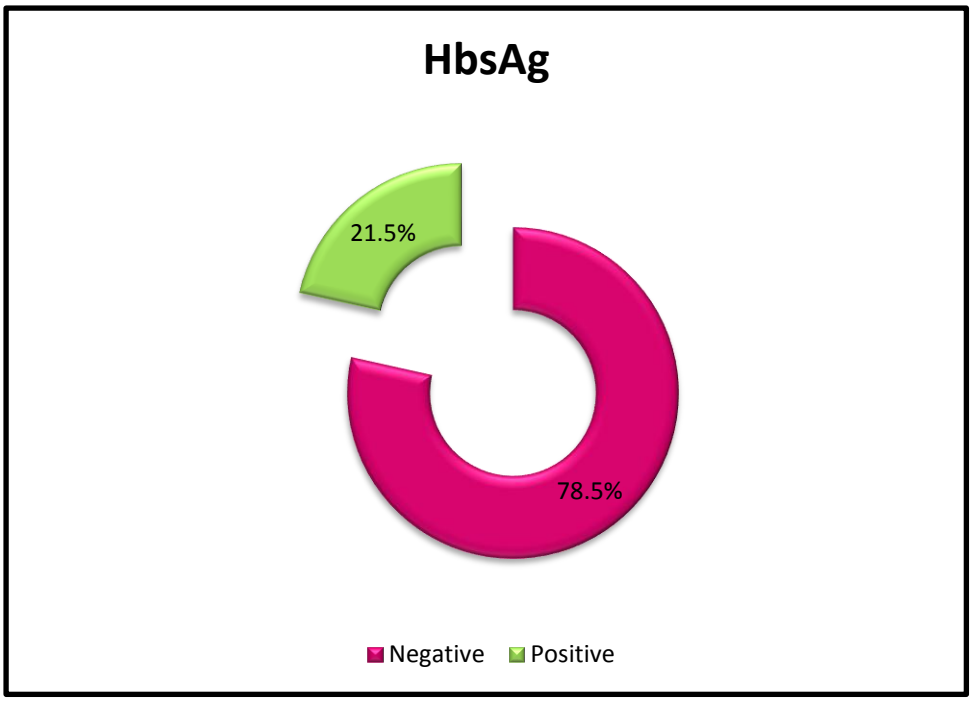


TABLE 12: LEPTOSPIROSIS

Leptospirosis	Frequency	Percent
Negative	77	97.5
Positive	2	2.5
Total	79	100.0

CHART 12: LEPTOSPIROSIS

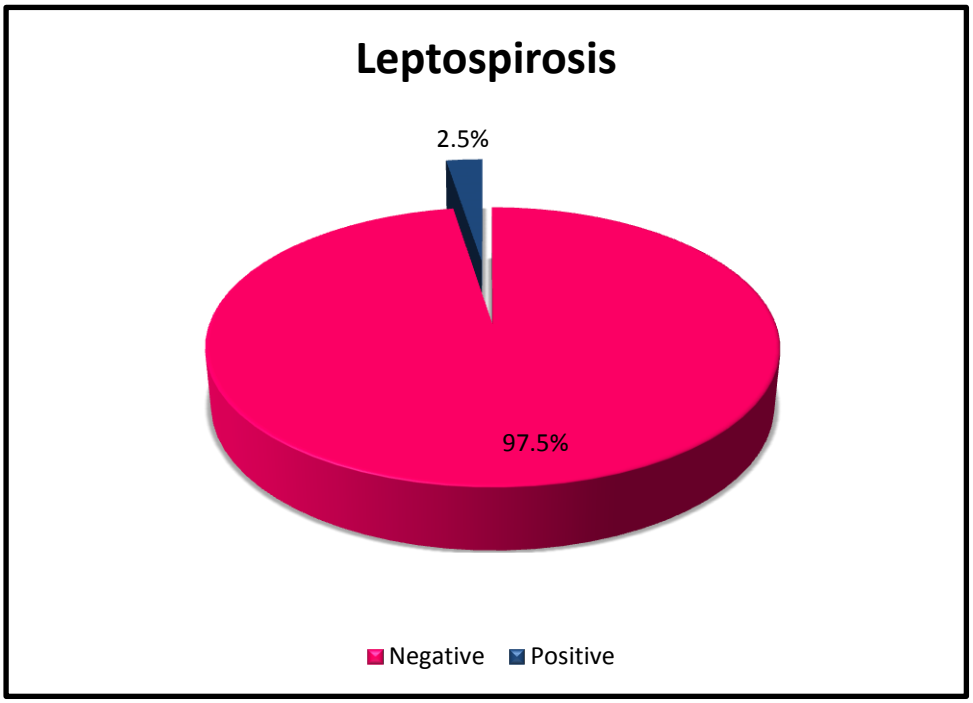


TABLE 13: SERUM BILRUBIN

Serum Bilirubin	Frequency	Percent
2 - 4	30	38.0
4 - 6	16	20.3
6 - 8	7	8.9
8 - 10	7	8.9
10 - 12	3	3.8
12 - 14	6	7.6
> 14	10	12.7
Total	79	100.0

CHART 13: SERUM BILRUBIN

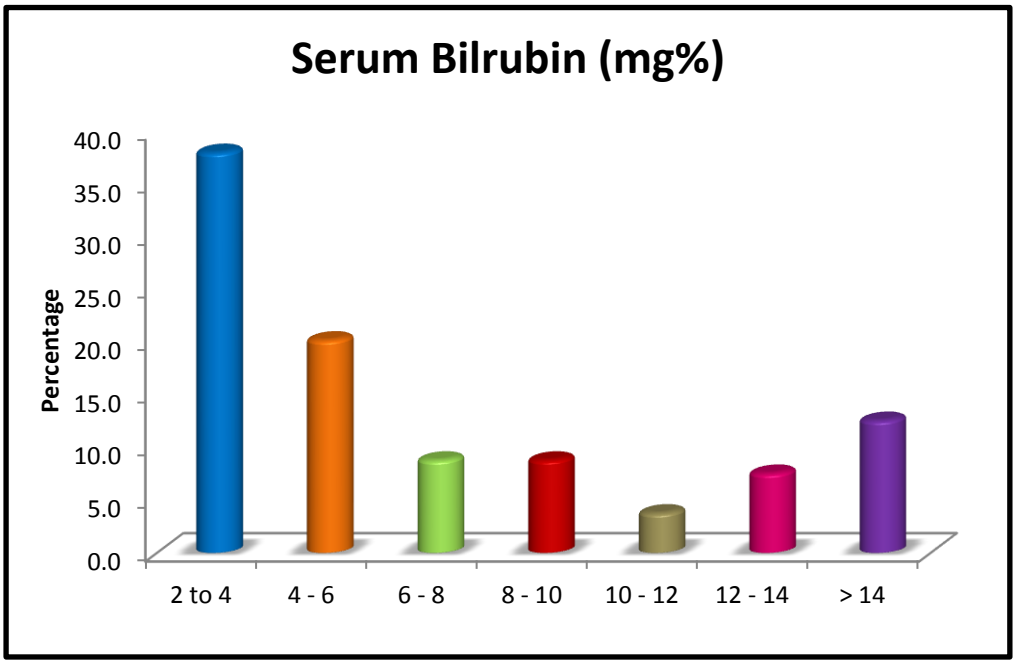


TABLE 14: DIAGNOSIS

Diagnosis	Frequency	Percent
1	47	59.5
1(B)	12	15.2
2	7	8.9
3	3	3.8
4	3	3.8
5	1	1.3
6	1	1.3
7	1	1.3
8	3	3.8
9	1	1.3
Total	79	100.0

CHART 14: DIAGNOSIS

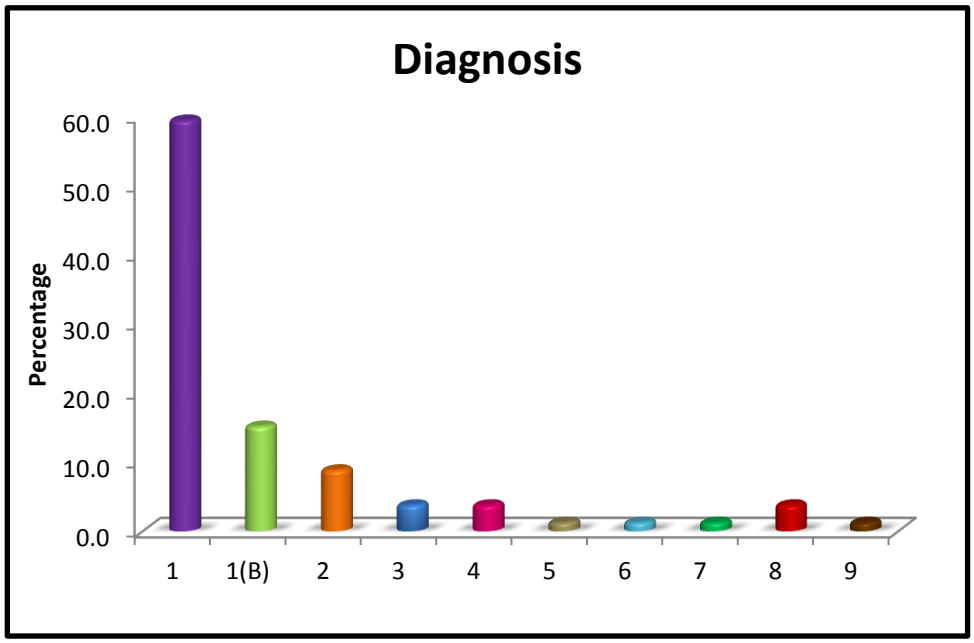


TABLE 15: BW

BW	Frequency	Percent
1 - 1.5	12	23.1
1.6 - 2	11	21.2
2.1 - 2.5	18	34.6
2.6 - 3	10	19.2
> 3.5	1	1.9
Total	52	100.0

CHART 15: BW

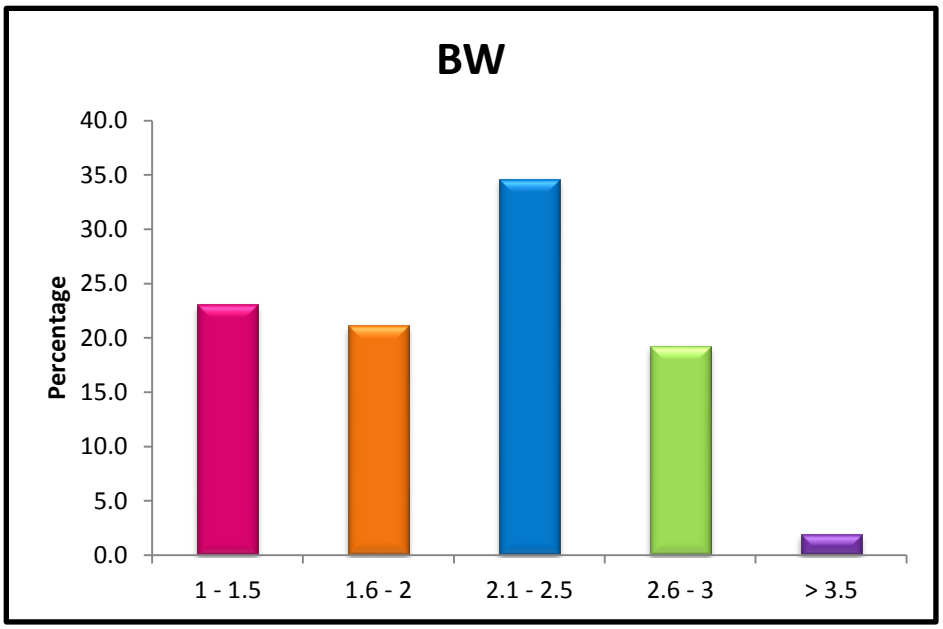


TABLE 16: MODE OF DELIVERY

Mode of Delivery	Frequency n	Percent %
Abortion	5	6.5
Discharge on Request and Lost Follow up	18	22.7
DIVC - Death	1	1.3
Forceps	1	1.3
Hepatic Coma - Death	1	1.3
HRF AND DEATH	2	2.5
LSCS	9	11.4
Normal	41	51.9
Total Abdominal Hysterectomy	1	1.3
Total	79	100.0

CHART 16: MODE OF DELIVERY

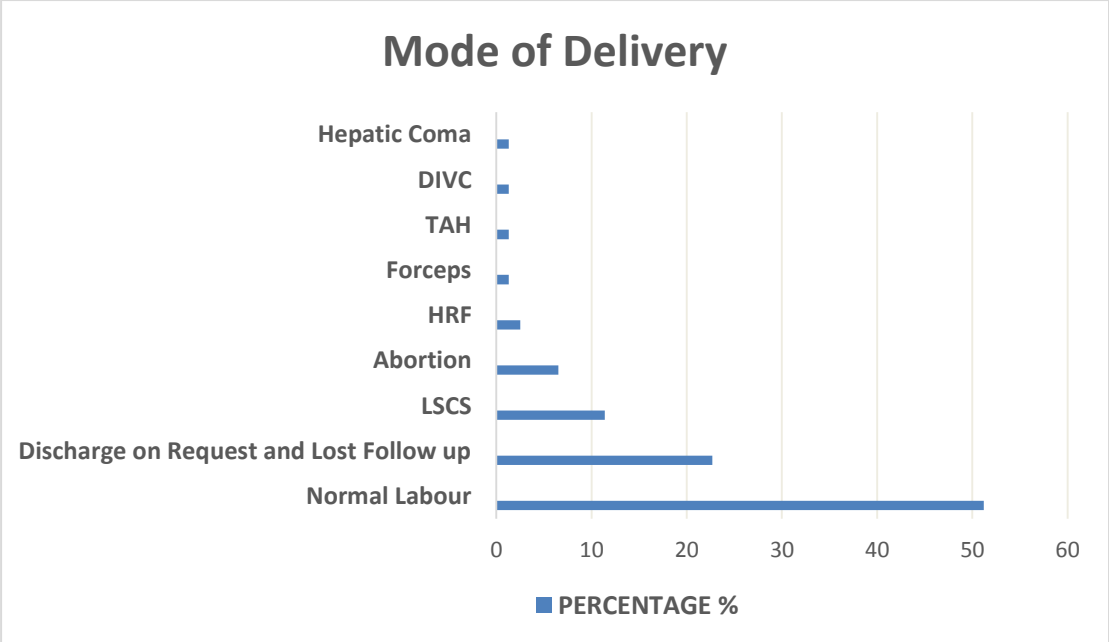


TABLE 17 : AGE WITH STATUS

Age	Status		Total
	Alive	Dead	
18 - 20 yrs	12	0	12
21 - 25 yrs	41	3	44
26 - 30 yrs	17	1	18
31 - 35 yrs	3	0	3
> 35 yrs	2	0	2
	75	4	79

CHART 17 : AGE WITH STATUS

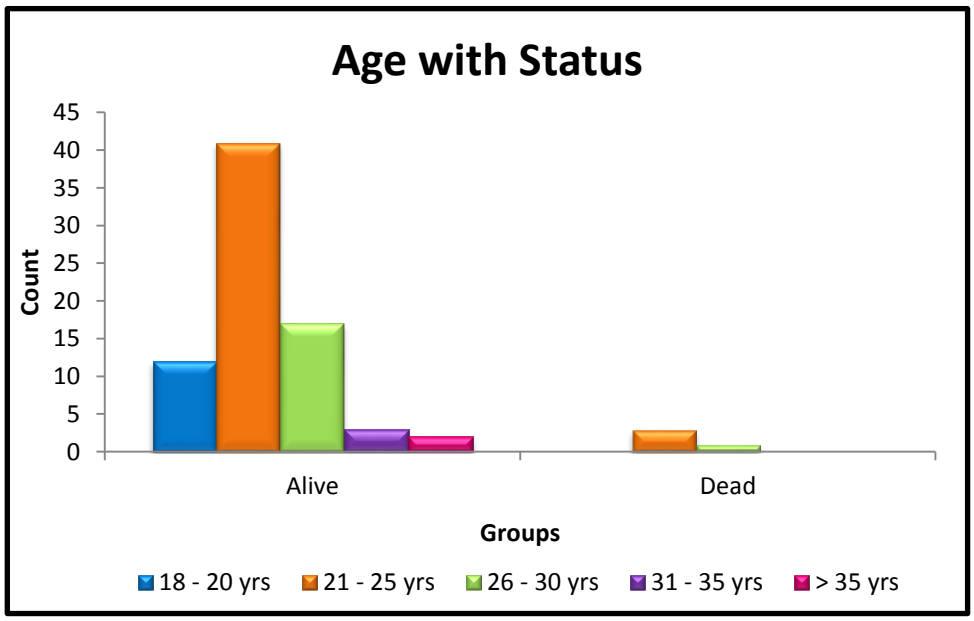


TABLE 18 : OBSTETRIC HISTORY WITH STATUS

Obstetric History	Status		Total
	Alive	Dead	
Multi	43	2	45
Primi	32	2	34
	75	4	79

CHART18 : OBSTETRIC HISTORY WITH STATUS

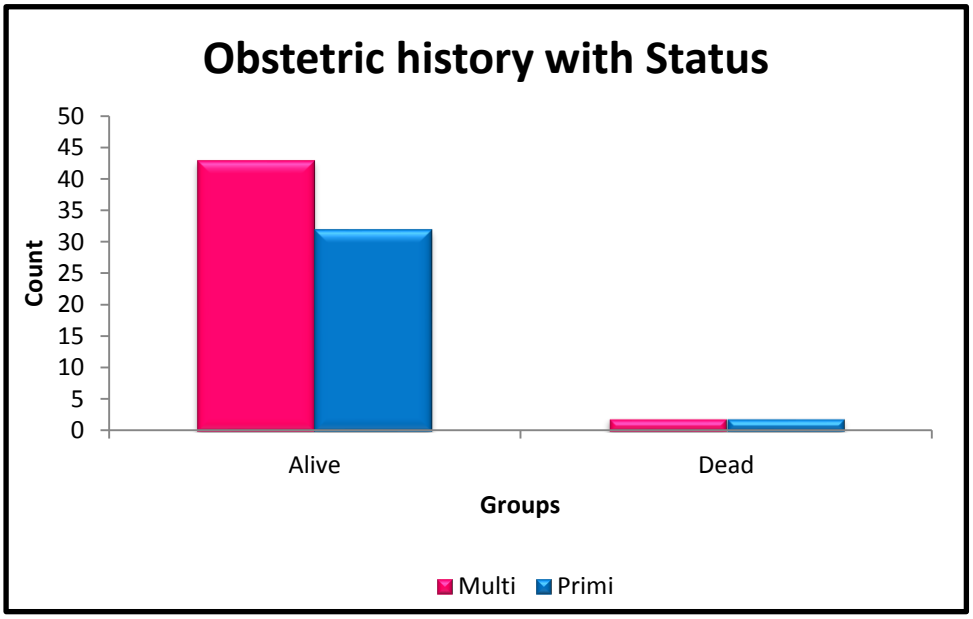


TABLE 19 : DURATION WITH STATUS

	Status		Total
	Alive	Dead	
Upto 5 days	25	3	28
6 - 10 days	41	1	42
11 - 30 days	5	0	5
> 30 days	4	0	4
	75	4	79

CHART 19: DURATION WITH STATUS

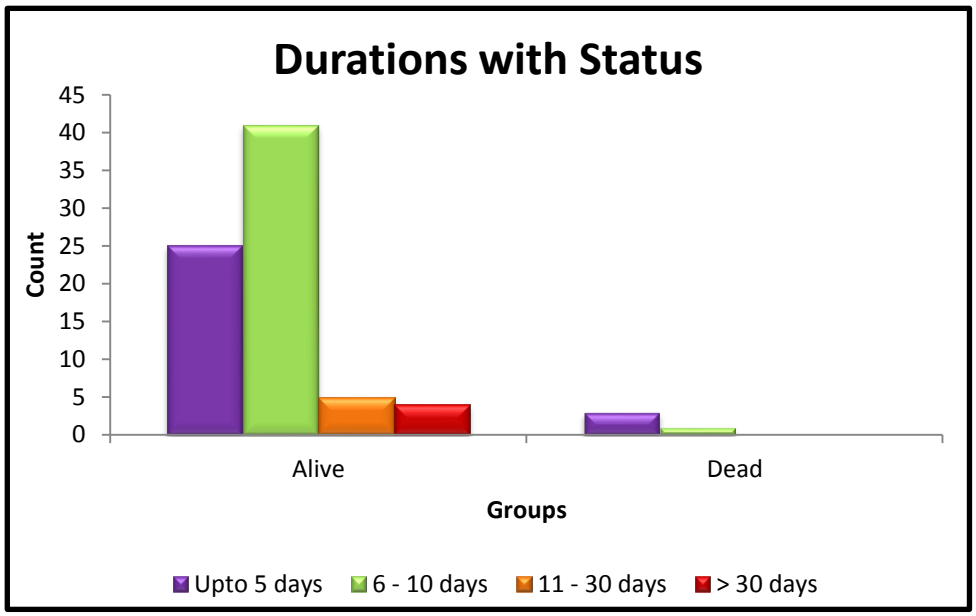


TABLE 20 : BS WITH STATUS

BS with status	Status		Total
	Alive	Dead	
Negative	3	1	4
Positive	72	3	75
	75	4	79

CHART 20 : BS WITH STATUS

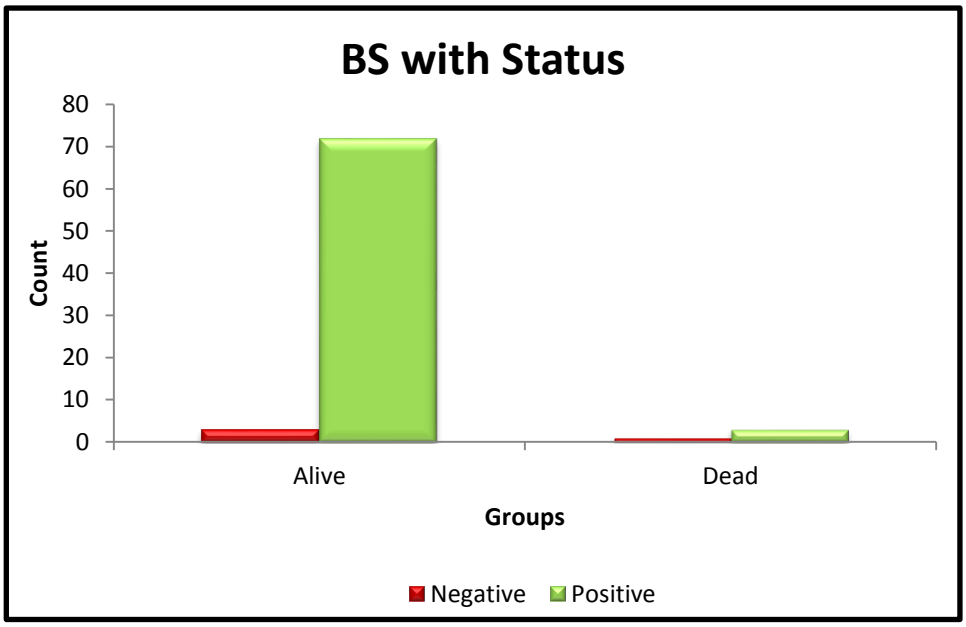


TABLE 21 : BP WITH STATUS

	Status		Total
	Alive	Dead	
Negative	3	1	4
Positive	72	3	75
	75	4	79

CHART 21 : BP WITH STATUS

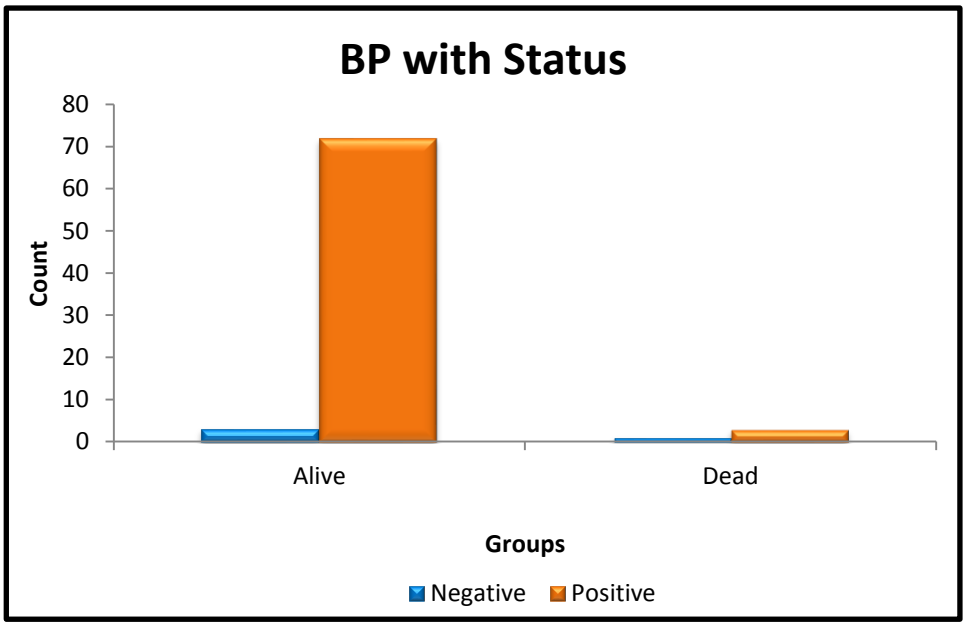


TABLE 22 : PROTEINURIA WITH STATUS

PROTEINURIA WITH STATUS	Status		Total
	Alive	Dead	
Negative	69	3	72
Positive	6	1	7
	75	4	79

CHART 22 : PROTEINURIA WITH STATUS

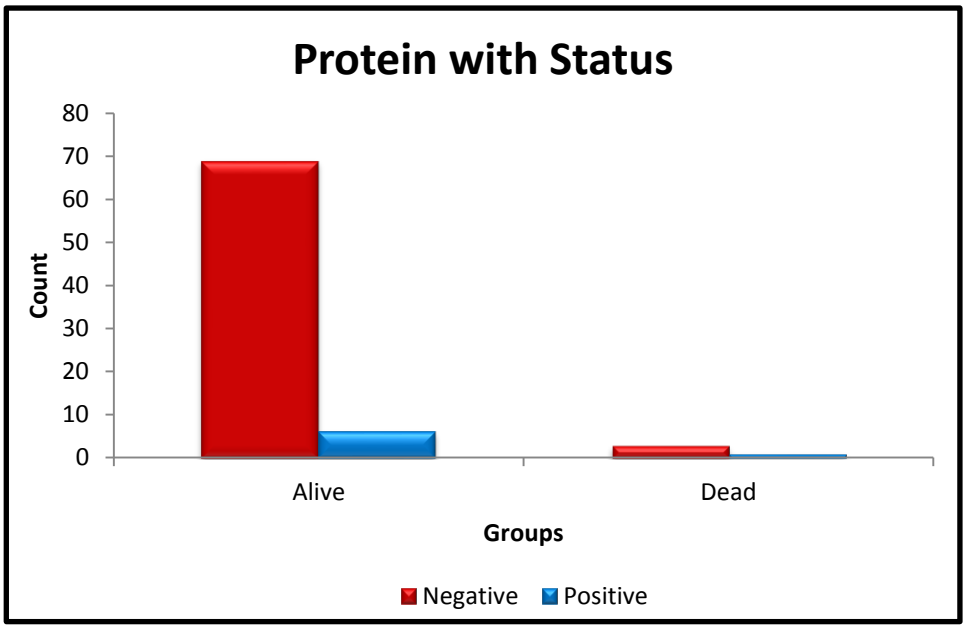


TABLE 23 : HbsAg WITH STATUS

HbsAg with status	Status		Total
	Alive	Dead	
Negative	59	3	62
Positive	16	1	17
	75	4	79

CHART 22 : HbsAg WITH STATUS

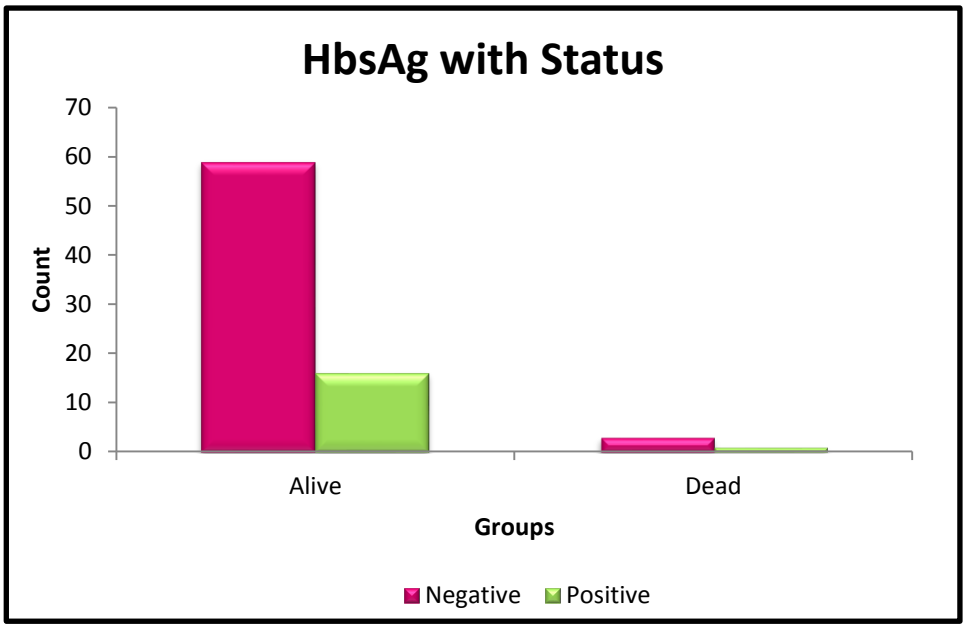


TABLE 24: LEPTOSPIROSIS WITH STATUS

	Status		Total
	Alive	Dead	
Negative	73	4	77
Positive	2	0	2
	75	4	79

CHART 24 : LEPTOSPIROSIS WITH STATUS

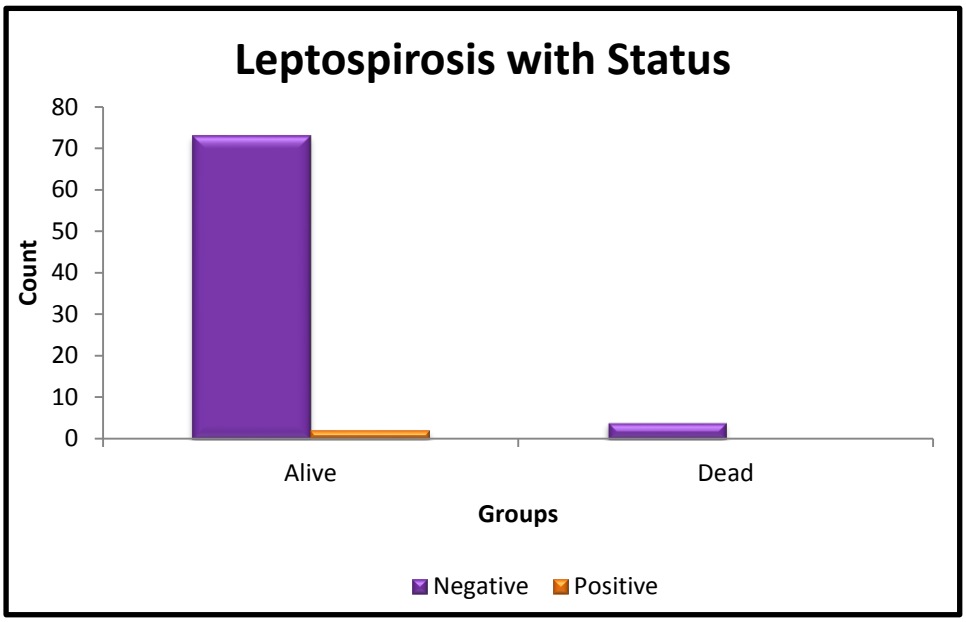
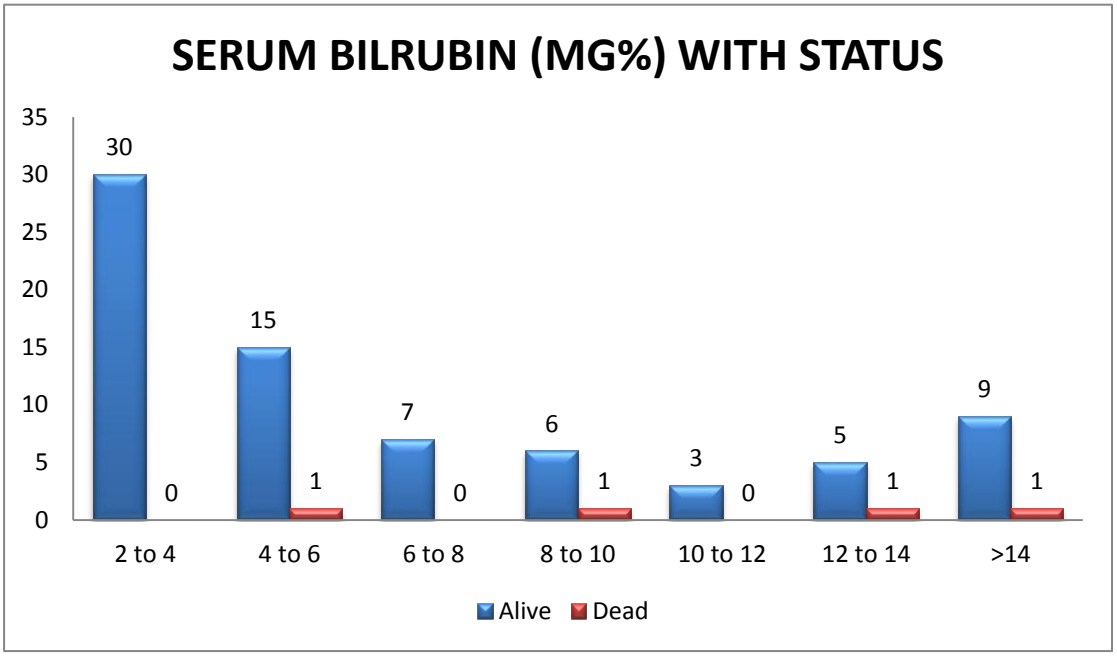


TABLE 25 : SERUM BILRUBIN (MG%) WITH STATUS

SERUM BILRUBIN (MG%) WITH STATUS	Status		Total
	Alive	Dead	
2 - 4	30	0	30
4 - 6	15	1	16
6 - 8	7	0	7
8 - 10	6	1	7
10 - 12	3	0	3
12 - 14	5	1	6
> 14	9	1	10
	75	4	79

CHART 25 : SERUM BILRUBIN (MG%) WITH STATUS


SERUM BILRUBIN (MG%) WITH STATUS
35
30
25
20
15
10
5
0
30
0
15
1
7
0
6
1
3
0
5
1
9
1
2 to 4
4 to 6
6 to 8
8 to 10
10 to 12
12 to 14
>14
Alive
Dead

OBSERVATION

- Of the 79 women studied,maximum(n=44,55.7 %) were in the 21-25 years age group followed by 22.8% in 26-30 years age group,15.2 % in 18-20 years age group,3.8 % in 31-35 years age group and 2.5 % in > 35 years age group.

- Of the 79 women studied 57 % were multiparous women and the rest 43 % were Primi women.

- High coloured urine was the most common symptom seen in 44.3% of the women. Second common symptoms were fever and Nausea & vomiting (40.9% each). Other common symptoms in decreasing order of frequency were loss of appetite(37.4%),upper abdominal (12.2 %),pruritis and clay coloured stools.

- Icterus was the most commonly elicited sign(60.9 %) followed by hepatomegaly(16.5 %),splenomegaly(6.1 %),Scratch marks(3.5 %) and ascites (0.9%).

- Out of the 79 patients, 40.5 % had complications:Complications were Anemia (25.4 %),pre-eclampsia(7.6%),hepatorenal failure(5.1%),Hemetemesis and encephalopathy(3.8 % each).

- The duration of symptoms were mostly between 6-10 days(53.2 %) followed by less than 5 days(35.4 %) , 11-30 days (6.3 %) and > 30 days (5.1 %).

LABORATORY PARAMETERS

URINE BILE SALTS

Urine bile salts was present in 94.9 % of patients and absent in 5.1 % of patients.

URINE BILE PIGMENTS

Urine bile pigments was present in 94.9 % of patients and absent in 5.1 % of patients.

URINE PROTEIN

Urine proteins was present in 8.9 % of patients and absent in 91.1 % of patients.

URINE SUGAR

Urine proteins was absent in all patients.

SERUM BILIRUBIN

The serum bilirubin levels of 38 % patients was between 2-4 mg/dl,20.3 % was between 4-6 mg/dl,8.9% was between 6-8 mg/dl, was between 8-10 mg/dl,3.8 % was between 10-12 mg/dl, 7.6 %was between 12-14 mg/dl,12.7 % was >14 mg/dl.

HEPATITIS B Antigen(HBs Ag)

HEPATITIS B Antigen was positive in 21.5 % patients and negative in 78.5 % patients.

M-FAT for leptospirosis was positive in 2.5 % patients and negative in 97.5 % patients.

Peripheral smear study and Widal test was normal in all patients.

MODE OF DELIVERY

Of the 79 women studied,

51.9 %(n=41) had normal vaginal delivery,

11.4%(n=9) had LSCS delivery,

6.4%(n=5) had abortion,

1.3%(n=1) delivered by forceps delivery,

1.3%(n=1) had total abdominal hysterectomy,

22.7%(n=18) discharged on request/lost follow up,

2.5%(n=2) expire because of hepatorenal failure,

1.3%(n=1)expired because of hepatic coma and DIVC each.

FETAL OUTCOME-BIRTH WEIGHT

Of the 79 patients,18 patients lost follow up.

Of the remaining 61 patients,5 had abortion and 4 patients expired.The remaining 52 patients delivered.

23 .1%(n=12) delivered babies having a birth weight between 1-1.5 kgs

21.2%(n=11) delivered babies having a birth weight between1.6-2.0 kgs

34.6%%(n=18) delivered babies having a birth weight between 2.1-2.5kgs

19.2%%(n=10) delivered babies having a birth weight between 2.6-3.0kgs

1.9%%(n=1) delivered babies having a birth weight above 3.0 kgs

DIAGNOSIS

Of the 79 patients cause of jaundice in

59.5% patients was diagnosed as viral hepatitis,

5.2% patients was diagnosed as Hepatitis B,

8.9% patients was diagnosed as HELLP syndrome,

3.8% patients were diagnosed each as Intrahepatic cholestasis of pregnancy, chronic liver disease with portal hypertension and leptospirosis,

1.3 % patients were diagnosed each as hyperemesis gravidarum,toxic hepatitis ,acute fatty liver of pregnancy and blood reaction.

DISCUSSION

Age: In the present study 41% of pregnant jaundiced women were between 21 and 25 years. This correlates with the study of Sheth Abhay et al., 1990, Devidner Kaur et al., 2001 and Kamala Jayaram et al., 1988. This age group is having the minimum fertility rate and maximum number of deliveries. Early age of marriage also contributes (I Western countries ony 20% are under 245 years)

Gravidity: In this study 45% of patient were multiigravida. It correlates with the study of Redi Rani et al., 1986 and J.S. Chauhan et al., 1983 (50%). Kamala Jayaram et al. 1986 and Devinder Kaur et al., 2001 had showed only 30% incidence among primigravida. Achar et al., and Sita Ratna et al., has also quoted higher incidence in multigravida and attributed this to nutritional factors.

1. Anorexia, nausea, vomiting and fatigue in anicteric forms may be confused with morning sickness.
2. Excess nutritional demand by the developing fetus in the late pregnancy.
3. Patients in early trimester seek native treatment.
4. Any illness in the third trimester is viewed seriously.

5. Jaundice may be exaggerated by the hormonal changes and impaired bilirubin secretion.

Socioeconomic Status: 95% of patients belonged to lower socio economic group in this study. This shows the influence of malnutrition, poor sanitation, water contamination, sexual abuse and poor health awareness in the development of jaundice as stressed by various authors.

Past History : 4 patients had contact with jaundiced patients, of which 2 of their husbands were HBsAg positive. These necessitates further serological analysis (Rohatagi et al., 1987). 2 patients of intrahepatic cholestasis had the past history of intense pruritis and progressive jaundice (from the third trimester and disappeared after delivery) in the previous pregnancy. It correlates with the study of Veena Agarwal, 2001. They need careful perinatal care.

Only 32% of patients were at the habit of drinking boiled water. Contamination of water and food left uncovered are the commonest source of infection for hepatits A and hepatitis E virus.

Etiology :

In the present study, viral hepatitis was found to be the commonest cause (59.5%) next being HELLP Syndrome (8.9%) third in order is chronic liver disease (3.8%) and infra hepatic cholestasis of pregnancy (3.8%). Other uncommon causes are Acute Fatty liver of pregnancy (1.3%) Leptospirosis (2.5%), Toxic hepatitis (1.3%), hyperemesis Gravidarum (1.3%) and blood reactions.

The study conducted by the Christian Medical College, Vellore, regarding the etiology of the disease shows:

Viral hepatitis : 11%	Intrahepatic cholestasis : 5%
Preeclampsia : 6%	Fatty liver of pregnancy : 3%
HELLP : 5%	Others : 70%

Symptoms : In the present study, yellow coloured urine and fever were the predominant symptoms. This correlates with the study of Subodh Singh R. Chauhan et al., 1991, but it contradicts the study of V.Issac, 1975 and Jai Bhagwan S harma et al., 1990 who had found more patients with gastrointestinal symptoms. The altered appetite may be mistaken for pregnancy symptom (J.S.Chaddha et al., 1983). Signs : In this study, 92% of patients presented with jaundice. This is comparable with the study of Kamala Jayaram et al., 1988 (93%). But Sunanda

Kulkarni, 1996 has reported jaundice only in 60% of cases and in 23% of cases liver was palpabale. Michael et al., 1951 has quoted as palpable liver as a good prognostic sign.

Biochemical Parameters : In the present study, 38% of maternal mortality were when the serum bilirubin was more than 10mg%. In the study of Chadda et al., (1983), majority of mortality occurred with serum bilirubin level more than 15mg% and high serum transaminate levels. 45.5% mortality were observed with serum transininase levels between 400 to 800 IU/L in the present study. Krisher, 1961 and C.M.Alwani has reported mortality with low levels and described this due to exhaustion by previous excessive release of serum transaminase (Issel Backer, 1987).

Management : Symptomatic and supportive therapy was given with bedrest, high carbohydrate diet, glucose and vitamins. The obstetric interventions were done only for HELLP syndrome. The facilities for freshblood transfusion were kept available.

Complications : 20 patients had anaemia; among them 7 had severe anaemia (<5gm%). and rest of them were between 7-10gm%. Among the 7, one had nutritional anaemia, one had anaemia because of abruptio

placenta and 5 had anaemia because of postpartum haemorrhage. Anaemia as an aggravating factor was studied by Sathyavathy et al., 1979. Among the 8 patients with HELLP Syndrome, one died

Pregnancy Outcome : Among the total 79 patients, 18 patients recovered and discharged and pregnancy outcome was not determined in them. 10% had abortion. 64% had preterm delivery and 26% had term delivery. This condradicts the study of Sheila Sherlock 1970 who has stated that viral hepatitis run the same course as in non pregnant women.

Thorling I, 1955 has reported only low preterm deliveries whereas Narayan 1969 made no such difference.

Increased incidence of abortion and preterm delivery may be due to high fever and the debilitating effect of viraemia of hepatitis.

Mode of Delivery : 11% of patients were delivered by LSCS for obstetric indications and 2% were delivered by forceps. 87% were delivered by sponteneous vaginal delivery, since they were preterm labour, labour was easier.

Early delivery by caesarean section to improve the maternal and fetal survival and arresting the disease is recommended by Peters et al., 1967 and Burroughs et al. 1983. However the risk of anaesthesia and those bleeding from operative site are factors against it. This view is supported by **Jai Bhagwan sharma et al., 1990, Reddy Rani et al., 1993** and Issac & Chandrasekar, 1945.

Maternal Outcome : Out of these 79 jaundiced pregnant women in this study, 4 women exprired, of which 1 patient died antenatally and 3 patients died following delivery or abortion.

55% of maternal mortality was found to be in those women between 20 and 24 years as comparable with the study of **Kamala Jayaram et al., 1988 (56%) and Sunanda Kulkarni, 1996 (52%).**

In the present study, primigravida constituted 48.10% of mortality. In the study of **Kamala Jayaram et al., 1988, 65%** were multigravida.

In the present study, only 27% of maternal mortality were in the III trimester. Increased mortality in the III trimester is supported by **Malkani, 1957 (75%), Naidu Viswanathan (41%), D'Cruiz et al.,**

1968 (81%), Seth Abhay et al., (1990) and Kamala Jayaram (1988).

In this study 64% of maternal mortality occurred postpartum. This is supported by the study of **Subodh Singh, R Chauhan et al., (1991) and Roy Chowdhary**. The coma is precipitated by stress and strain of labour.

In the present study 81.8% of patients died within 5 days of onset of symptoms and 18.2% died within 10 days of onset of symptoms. This is comparable with the study of Sathyavathy, 1979 and **Sunanda Kulkarni, 1996.**

1 patient died of HELLP syndrome, one patient (9%) died of blood reaction, and one patient died of Leptospirosis.

The postpartum haemorrhage is common because of uterine atony which is due to debilitating illness and coagulation failure.

Two patients following delivery rapidly progressed to hepatic coma and died. The stress and strain of labour might have precipitated hepatic coma.

One patient developed coagulation failure, haemetemesis and malena and died. The liver synthesises the coagulation factors II, V, VII, IX & X. In acute fulminant hepatitis, their synthesis is impaired thus precipitating coagulation failure. Haemetemesis, malena and uraemia favour the production of nitrogenous substances. Because of hepatic failure, they are shunted from the portal circulation directly to the systemic circulation and to the brain and precipitate coma.

One antenatal jaundiced woman developed hepatic coma and died probably because of sepsis.

Causes of Maternal Mortality by various authors

1. **Hepatic coma** -	**Varner 1980 75%**
	Scully et al., (1981) - 80%
	Chaddha et al., (1983) - 43%
	Kamala Jayaram et al., (1981) - 73%
	Sunanda Kulkarni (1996) - 60%
	Devinder Kaur et al., (2001) - 26.7%
2.Postpartum Haemorrhage -	**Chaddha et al., (1981) - 10.5%**
	Kamala Jayaram et al., (1988) - 14%
	Jai Bhagwan Sharma et al., (1990) -69%

Sunanda Kulkarni (1996) - 13%

Devinder Kaur et al., (2001) - 26.7%

3. Hepatorenal failure - **Chaddha et al., (1983) - 6.6%**

Kamala Jayaram et al., (1988) - 1.7%

Sunanda Kulkarni (1996) - 13.04%

4. Coagulation failure - **Chadda et al., (1983) - 1.3%**

Kamala Jayaram (1988) - 10%

In our study, there were two cases of suspected acute fatty liver of pregnancy. They presented in the III trimester with jaundice. One patient had mild renal failure, other patient had mild bleeding tendency with prolongation of the clotting time (20-25 minutes).

Immediately after admission both of them delivered preterm dead fetuses. Both of them recovered after delivery with intensive care therapy and blood transfusion.

In the study conducted by **George M.Chandy et al., Christian Medical College, Vellore, 2001**, they have reported 18 cases of acute fatty liver of pregnancy from January 2018 to February 2019. Among them 2 died - one because of multiple organ failure and other because of disseminated intravascular coagulation.

All the other patients survived because of the team work by the senior obstetricians, gastroenterologist, nephrologist, anaesthetist and intensive care therapy.

Fetal outcome : Among the 15 term babies, one was still born. Among the 37 preterm deliveries (2 twin deliveries), one was IUD, 8 was still born and 10 neonatal mortality. All the neonatal des were because of prematurity and low birth weight.

Prematurity and low birth weight as a cause of neonatal mortality was stressed by various authors (Jai Bhagwan Singh, 1990, Subodh Singh et al., 1991, Devinder Kaur et al., 2001). Narayan (1969) had a large still birth in his series. Thorling I (1955) had low incidence of prematurity.

Fetal loss by abortion and prematurity may be due to high fever and general debility associated with high viraemia. Prabhu and Pendarkar, 1984 have demonstrated immunological changes in the placenta. The fetal immunological system developing in the second half of pregnancy may react to the virus or virus particle to bring about placental damage by forming fetal antibodies. Since the fetal antibodies

are produced only after 20 weeks, preterm delivery is the common sequalae **(Sheth Abhay, 1990).**

Among the 3 cases of intrahepatic cholestasis, 2 gave birth to preterm babies one gave birth to term baby, and the three survived. Among the 2 cases of acure fatty liver of pregnancy both the babies were still born.

Fetal anomalies : There was no congehital anomaly in the present study. Shah had reported that there is no convincing evidence that jaundice in the first trimester can cause fetal anomalies. So jaundice is not an indication for medical termination of pregnancies.

Placental abnormality : Macroscopically there were no anomalies. Microscopic examination was not done. Naidu and Viswanathan had reported bile stained amniotic fluid and membranes in their study.

Development of Neonatal Jaundice : 4 babies had only physiological jaundice. A jaundiced mother giving birth to a jaundiced baby is rare because in most cases of jaundice (viral hepatitis and cholestasis), conjugated bilirubin is present to which placenta is impermeable (Sechar). The reported incidence by various studies is 1.08%.

Transplacental transmission of HBsAg : The cord blood of the 7 babies of HBsAg positive mothers were tested for HBsAg, but all of them were negative for HBsAg Similar study was conducted by Okado. All the babies received immunoglobin and vaccination.

SUMMARY

1. The study was undertaken in the Institute of Obstetrics and Gynaecology Government Hospital for Women and Children, Egmore, Chennai from January 2018 to February 2019. 79 patients had jaundice which constitutes the incidence of 1.42 per 1000 population.

2. Viral hepatitis is the commonest cause.

3. Out of these 57 cases of viral hepatitis, 17 were positive for HBsAg.

4. Jaundice in pregnancy has a predilection to young women (21-25 years). Fulminant hepatic failure is also higher in this age group.

5. Jaundice in pregnancy had a higher predilection in multigravida. .

6. The maximum incidence was in the third trimester.

7. The mortality was more in the puerperium.

8. These people belonged to lower socioeconomic group.

9. Past history of jaundice was evidenced in 2 cases of intrahepatic

cholestasis, 4 patients had contact with jaundice.

10. Mortality rate was high in the individuals with acute onset and rapid progression of jaundice.

11. Etiological diagnosis was supported by clinical features, laboratory investigation, ultrasonogram and serological marker.

12. Anaemia and PIH in a pregnant jaundiced women worsens the prognosis.

13. Development of hyperpyrexia, bleeding manifestations, generalised edema, ascites, shrinkage of liver, hepatorenal syndrome and coma heralded the worst prognosis.

14. Twin pregnancy was found to have very bad maternal and fetal prognosis.

15. We had 3 cases of intrahepatic cholestasis, 2 had preterm delivery, one had term delivery. All the 3 improved clinically immediately after delivery.

16. We had 2 cases of suspected acute fatty liver of pregnancy, one had mild renal failure and one had bleeding tendency; both delivered spontaneously immediately after admission. Both babies were still born. Both the patients recovered postnatally following intensive care therapy and blood transfusion.

17. Maternal and fetal prognosis were found to be very bad at very high levels of serum bilirubin, rapidly rising and persisting at very high levels without fall, but prognosis was affected by low levels also.

18. Serum alkaline phosphatase levels were raised to high levels in intrahepatic and extrahepatic cholestasis. It did not correlate with the prognosis.

19. Very high levels of serum transaminase indicate marked hepatocellular damage and showed bad prognosis. Sudden fall to low level is also ominous.

20. In a jaundiced pregnant woman, persistent severe hypoglycemia, severe hypoproteinemia with marked reversal of Albumin, globulin ratio, marked rise in blood urea and serum creatinine, serum uric acid > 7 mg and severe leucopenia are ominous signs.

21. Serum prothrombin time is said to be a good indicator of prognosis.

22. No significance could be given to the mode of delivery in relation to maternal and fetal outcome.

23. The stress of labour predisposes the women to coma. So careful observation is a must in the immediate pospartum period.

24. Low birth weight due to prematurity predisoposed to high fetal loss. No sex predilection was found among the babies.

25. Incidence of preterm labour was high in our study.

26. Perinatal mortality rate is 37.03 per 1000 live birth.

27. No congenital anomalies was detected in the babies.

28. No placental anomalies detected.

29. No case of pathological neonatal jaundice in this study.

30. Maternal mortality among the jaundiced patients is 5.06%.

31. Viral hepatitis contributes maximum to the incidence and mortality. Two Maternal deaths were due to Hepatitis B viral infection.

32. Fulminant hepatic failure with hepatic coma, hepatorenal syndrome, postpartum haemorrhage and coagulation failure were the main causes of maternal mortality.

33. No cord blood was positive for HBsAg in the new born babies of HBsAg positive mothers. All received passive and active immunisation.

CONCLUSION

The development of jaundice during pregnancy is an important health hazard and needs careful monitoring during antepartum, peripartum and postpartum period.

Even though the course of the disease can not be altered by early detection of the the aravating factors can be avoided by the obstetrician by careful monitoring. disease, gg Jaundice due to acute hepatitis needs only supportive therapy. Though the outcome of hepatitis is based on the immunity of an individual, in pregnancy it is decided by many factors like the general health of the patient and obstetrical problems like anaemia, pregnancy induced hypertension and postpartum haemorrhage.

As prevention is better than cure, primary prevention by various health programmes and health education to pregnant women will help to live in a healthy environment. This can be achieved with the help of paramedical staff and mass media.

Use of disposable syringes and needles and careful screening of blood for many complications. serological markers before blood transfusion will help in voiding many complications.

All the pregnant mothers should be screened for HBsAg as a routine at primary, secondary & tertiary centres. Immunoprophylaxis should be given to the exposed pregnant women. To reduce the incidence of hepatitis B infection at the root level, immunoprophylaxis should be given to the newborns of the symptomatic and asymptomatic carrier mothers.

All the primary centres should make early referral of complicated pregnancies. We have to anticipate complications like hepatic encephalopathy and coagulation failure and check the availability of injection Vitamin K, fresh blood and fresh frozen plasm whenever we are having cases of jaundice complicating pregnancy.

At the secondary and tertiary level a team work by obstetricians, gastroenterologist, neurologist, nephrologists, microbiologist will help to achieve good outcome in jaundiced patients during antenatal, perinatal and neonatal period.

BIBLIOGRAPHY

1. Nor The Medical clinics of North America July 1989 Page No: 1041-1047.
2. The Medical Clinics of North America, September 1996. Page No.1167 to 1186.
3. Clinics in Liver Disease Caroline A Riely, MD, Vol.3 February 1999. Williams Obstetrics - 21st Edition. Chapter 48 Page No.1283 to 1296.
4. Davidsons Principles and Practice of Medicine - Eighteenth Edition. Page NO. 683 to 736.
5. Harrison's Text Book of Medicine 15th Edition Page No: 1743 to 1770. Liver Disease - Diagnosis and Management : Bruce R.Bacon, M.D. and Adrian M.D. 13isceglie, M.D. Page No: 344 to 358.
6. Oxford Textbook of Clinical Hepathology (1991) volume - 2 Page No: 868 to 1285.

7. Oxford Textbook of Medicine (1996) Third Edition - Volume 2 Page No.2054 to 2060.
8. Oxford Textbook of Clinical Hepatology - second Edition Volume 2 Page No.1364 to 1913.
9. Diseases of the Liver and Biliary System - Sheila Sherlock - Ninth Edition. Page No. 452 to 459.
10. Schiff's Disease of the liver - Eight Edition. Volume 2 page NO.1337 to 1355.
11. Liver Disease in Pregnancy Published in American Family Physician, February 1999 by the American Academy of Family Physicians. Page 1 to 9.
12. Samuels P. Cohen AW Pregnancies complicated by Liver Disease and liver dysfunction. Obstetrics and Gynaecology Clinics of North America. 1992 December Page 745 to 763.
13. The Merck Manual Section 18. Chapter 251 - 'Pregnancy Complicated by Disease - Hepatic Disorder.

14. Jaundice in Pregnancy by Diaa. M.EI Mowafi, M.D. (http://matweb.hcuge.ch/E/Mowafi).

15. Infectious Diseases of the female Genital Tract Richard L.Swelt

16. Medical disorders complicating pregnancy. Hon: Mother and child glossary.

17. J.S. Chaddha & M.Y. Raval - infective hepatitis and pregnancy. The Journal of obstetrics and Gynaecology of India. April 1983 - Page 214 - 217.

18. A.K. Bey et al - Hepatitis .B Virus Infection in Pregnancy. Its effects on mother and Foetus. The Journal of Obstetrics and Gynecology of India. 1983 Page No: 996 to 999.

19. (Mrs.) C.M. Alwani et al - Pregnancy Associated with infective Hepatitis - The Journal of Obstetrics and Gynecology of India - 1983 Page 677 to 680.

20. C.V. Hedge & C.M. Alwani - A review of Infective hepatitis in pregnancy - The cur Journal of Obstetrics and Gynaecology of India - 1984 Page No: 1120 to 1126.

21. S.Sasikala and K. Chandrasekar - Infective Hepatitis in pregnancy - The Role of exchange transfusion in hepatic failure - The Journal of Obstetrics and Gynaecology of India - June 1986. Page No: 441 to 444.

22. S.K.Sachdevi et al; Prevalence of Hepatitis B surface antigen in pregnant women from Delhi. - The Journal of Obstetrics and Gynaecology of India June 1986, Page 445 to 447.

23. P.Rohatagi et al., The role of Hepatitis '13' surface Antigen (HBsAg) during pregnancy with or without jaundice in vertical transmission in the new born - The Journal of Obstetrics and Gynaecology. August 1987 Page No: 530 to 533.

24. V.Kamalajayaram et al., A study of Maternal Mortality in Jaundice - The Journal of Obstetrics and Gynaecology. August 1988 Page No: 439 to 441.

25. Jai Bhagwan Sharma et al., Pregnancy outcome in Hepatic Coma. The Journal of Obstetrics and Gynaecology of India - August 1990, Page No.522 to 525.

26. Sheth Abhay et al. Prospective study of 146 cases of Acute viral Hepatitis in pregnancy. The Journal of Obsetrics and Gynaecology of India. June 1990, Page No: 340 to 344.

27. Subodhsingh R.Chauhan et al - Jaundice in Pregnancy. The Journal of Obstetrics and Gynaecology of India. April 1991. Page No: 187 to 189.

28. Reddi Rani Et al. Jaundice in Pregnancy. The Journal of Obstetrics and Gynaecology of India. August 1993 Page No: 532 to 535.

29. Sunanda Kulkarni A five year study of Maternal Mortality in Jaundice. The Journal of Obstetrics and Gynaecology of India. August 1996 Page No: 556 to 561.

ABBREVIATIONS

Headings:

OH – Obstetric History
WOG – Week of Gestation
GPL – Gravida, Para, Live
BS – Bile Salts
BP – Bile Pigment
HB – Haemoglobin
P. Smear – Peripheral Smear

Complications:

Anaemia
Preeclampsia
Bleeding Tendency
Hepatorenal failue (HRF)
Hepatic coma
PPH – Postpartum Haemorrhage
DIVC – Disseminated Intravascular Coagulation

Symptoms

High Coloured Urine
History of Fever
Loss of Appetite
Nausea and Vomitting
Abdominal Pain
Itching
Pruritis
Clay Stools
Loose Stools
Abdominal distension

Diagnosis

Viral Hepatitis
Hepatitis B virus infection
HELLP syndrome
Intrahepatic cholestasis of pregnancy
Chronic liver diseases with portal hypertension
Hyperemesis gravidarum
Toxic Hepatitis
Acute fatty liver of pregnancy
Leptospirosis
Blood reaction

Signs

Jaundice
Hepatomegaly
Spleenomegaly
Ascites
Scratch Marks

Pregnancy Outcome

Normal – Normal Vaginal Delivery
ND – Neonatal Death

INFORMATION SHEET

- We are conducting a study on **"Jaundice Complicating Pregnancy – Maternal and Foetal Outcome"** among patients attending Institute of obstetrics and gynaecology, Chennai and for that your clinical details may be valuable to us.

- We are selecting certain patients and if you are found eligible, we may be using your clinical details in such a way so as to not affect your final report or management.

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date: .

CONSENT FORM

STUDY TITLE: **Jaundice Complicating Pregnancy – Maternal and Foetal Outcome**

STUDY CENTRE : **Institute of Obstetrics and Gynaecology**
Madras Medical College,
Chennai.

PARTICIPANT NAME : AGE: SEX:
MRD.NO:

I confirm that I have understood the purpose of study, I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the procedure, I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it. Even if I withdraw from the study, I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any or results that arise from the study.

I hereby consent to participate in this study of "Jaundice Complicating Pregnancy – Maternal and Foetal Outcome "

Signature of Investigator: **Place :**
Date

Study Investigator:
Signature / Thumb Impression of patient

INFORMED CONSENT FORM

STUDY PLACE: Institute of Obstetrics and Gynaecology

TITLE OF THE STUDY: **Jaundice Complicating Pregnancy – Maternal and Foetal Outcome**

NAME OF THE INVESTIGATOR : Dr. SARANYA. G

NAME OF THE PARTICIPANT: AGE: SEX:

HOSPITAL NUMBER:

1. I have read and understood this consent form and the information provided to me regarding the participation in the study.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator
5. I have been advised about the risks associated with my participation in this study.
6. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.
7. I have not participated in any research study in the past.
8. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
9. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
10. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
11. I have understand that my identity will be kept confidential if my data are publicly presented
12. I have had my questions answered to my satisfaction.

13. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Name and signature / thumb impression of the participant

Name _____ Signature_____

Date_____

Name and Signature of impartial witness:

Name _____ Signature_____

Date_____

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature_____

Date_____

PROFORMA

Unit :

Date of Admission

Date of Discharge:

Patient's Name :

Age : Religion: Hindu/Muslim

I.P. No. : Christian/Others

Address :

Socio Economic Status

GRAVIDA PARA LIVE BIRTH ABORTION LMP:

EDD:

Symptoms :

1. Onset of Symptoms : Acute/Insidious
2. Duration of present illness :
3. Interval between onset of illness and coma
4. Symptoms with duration
    a. Loss of appetite
    b. Nausea and vomiting
    c. Fever
    d. Headache / Body Pain
    e. Abdominal Discomfort/ Pain
    f. High Coloured Urine
    g. Itching
    h. Pruritis
    i. Stools: Normal/Pale
    j. Bleeding per vaginum
    k. Draining per vaginum
    l. Labour pains

Obstetric History

1. Previous IUD, still birth, abortion
2. Any pregnancy complications

Past History

H/O jaundice in the previous pregnancies

H/O contact with jaundiced patients

H/O jaundice in the past

H/O blood transfusion

H/O multiple injections

H/O Drug intake

Habit of Drinking boiled water

Family History of Jaundice

Personal Historyof Exposure

General Examination

1. Conscious/stuperous
2. Nourishment and built
3. Anaemia
4. Jaundice
5. Edema
6. Palmar erythema
7. Spider Angioma
8. Purpura
9. Clubbing and Cyanosis
10. Lymphadenopathy
11. Flapping tremor
12. Foetor hepaticus
13. Bleeding manifestations

Vital Signs

Pulse

Temperature

Respiratory Rate:

Blood Pressure

Cardiovascular System : JVP

Heart Sounds/Murmur

Respiratory System

Central Nervous Systems

1. Level of consciousness
2. Orientation
3. Pupils: Normal/Dilated/Constricted Reaction to Light
4. Reflexes : Plantar reflex
   Deep reflexes: Normal/Absent/ sluggish/Exaggerated
5. Cerebellar Signs

Abdominal Examination

Liver : Normal / Enlarged: Soft/Firm/ Hard/smooth/nodular/tender/non tender

Gall bladder : Tender/Non Tender

Spleen : cm; soft/firm

Free Fluid : Present/Absent

Uterus size : corresponds to Weeks

Contractions

Presentation

Position: Engaged /Unengaged

FHR:

Liquor amount

CPD Assessment

Per vaginal examination

Investigation

1. Blood Grouping and typing
2. Urine Bile Salts, Bile pigments, protein, sugar microscope
3. Stool: Cyst and Ova
4. Complete Haemogaram:

   Haemoglobin Bleeding Time

   Total count : Clotting Time

   Differential count :

Periphereal Smear : Malarial parasites

Fragmented RBC's

Type of anaemia

5. Blood Urea — Serum ceratinine
   Blood Sugar — Serum electrolyte
   Serum fibrinogen — Sr. Uric Acid

6. Liver function tests

   Serum bilirubin — Total:

   Direct :

   SGOT : SGPT:

   Serum Alkaline phosphatase:

   Serum proteins : Total

   Albumin/Globulin Ratio

   Serum for leptospirosis

   VDRL

   HBsAg screening

   Widal test

Management

1. Diet
2. Vitamins ./ Vitamin K
3. Antipyretics
4. Antibiotics
5. Anticonvulsants
6. Stomach wash and Bowel wash
7. Lactisyn syprup

8. Blood Transfusion and fresh frozen plasma
9. Laour
10. Normal Vaginal forceps delivery/LSCS
    a. Duration of Labour : I stage
                            II stage
                            III stage

    Induction/Acceleration

    Complications

Results

1. Abortion
2. Intrauterine death
3. Still birth
4. Preterm delivery

    Baby alive Neonatal death
5. Mother pregnancy continued Recovered/Expired

    Pregnancy terminated/delivered Recovered/Expired
6. Baby
    a. Pre term/Term/Post term
    b. Sex
    c. APGAR 1 mt : 5 min
    d. Birth weight
    e. Congenital anomalies
    f. Discharged alive
    g. HBsAg in cord blood

Placental abnormality

Cord abnormality

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr. G. SARANYA
I Yr. MS PG in OBSTETRICS & GYNAECOLOGY
IOG/MMC
CHENNAI
Dear Dr.G. SARANYA

The Institutional Ethics Committee has considered your request and approved your study titled **"JAUNDICE COMPLICATING PREGNANCY – MATERNAL AND FOETAL OUTCOME" - NO.33032018**

The following members of Ethics Committee were present in the meeting held on **13.03.2018** conducted at Madras Medical College, Chennai 3

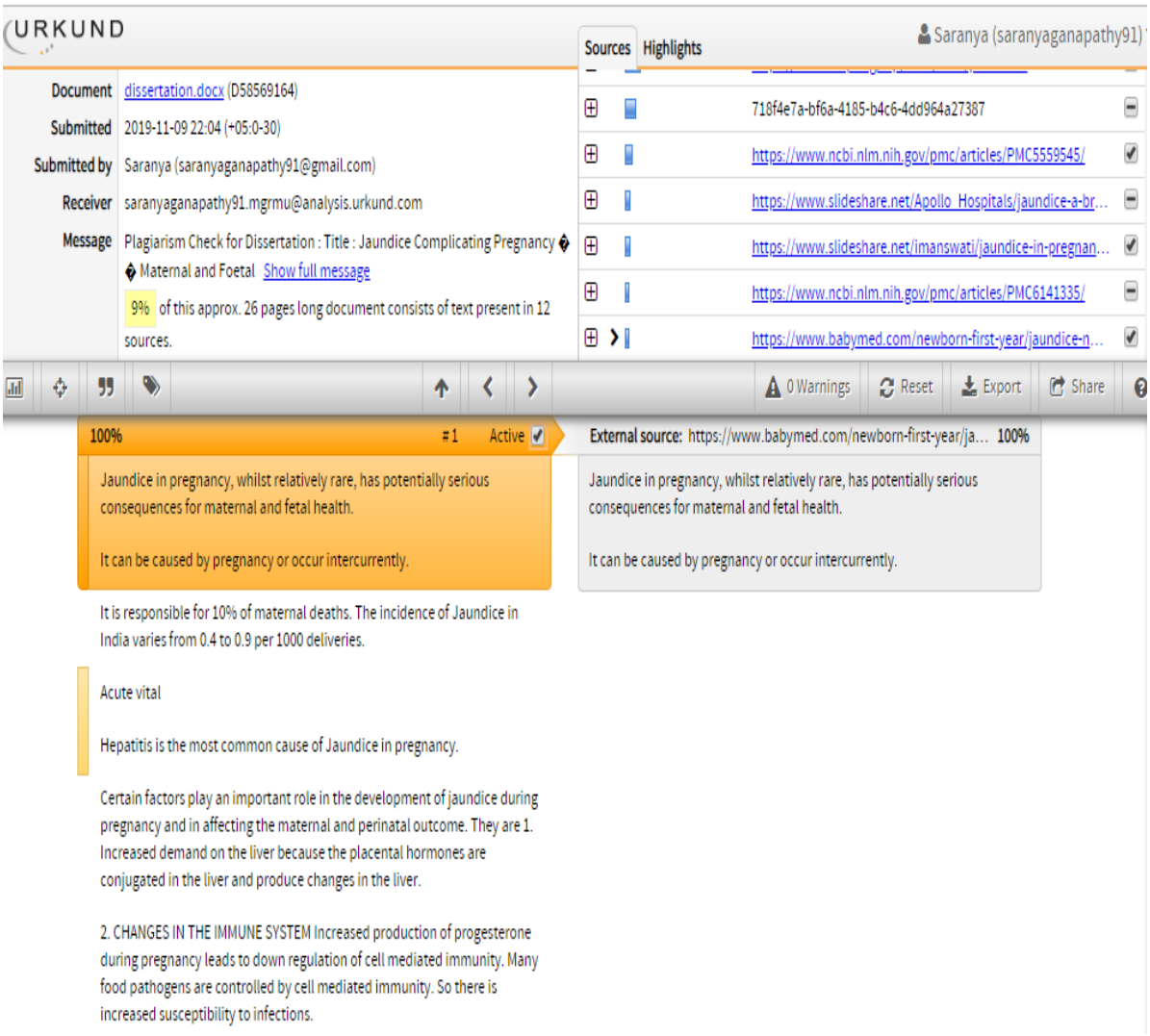
1. Prof.P.V.Jayashankar	:Chairperson
2. Prof.R.Jayanthi,MD.,FRCP(Glasg) Dean,MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC	: Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH	: Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
9. Prof. S. Purushothaman, Associate Professor of Pharmacology, MMC,Ch-3	: Member
10.Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3	: Member
11.Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3	: Member
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist
14.Thiru K.Ranjith, Ch- 91	: Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

19/3

Member Secretary – Ethics Committee



It is responsible for 10% of maternal deaths. The incidence of Jaundice in India varies from 0.4 to 0.9 per 1000 deliveries.

Acute vital

Hepatitis is the most common cause of Jaundice in pregnancy.

Certain factors play an important role in the development of jaundice during pregnancy and in affecting the maternal and perinatal outcome. They are 1. Increased demand on the liver because the placental hormones are conjugated in the liver and produce changes in the liver.

2. CHANGES IN THE IMMUNE SYSTEM Increased production of progesterone during pregnancy leads to down regulation of cell mediated immunity. Many food pathogens are controlled by cell mediated immunity. So there is increased susceptibility to infections.

URKUND

Urkund Analysis Result

Analysed Document:	dissertation.docx (D58569164)
Submitted:	11/9/2019 5:34:00 PM
Submitted By:	saranyaganapathy91@gmail.com
Significance:	9 %

Sources included in the report:

Dissertation P.docx (D31290808)
dissertation before pliagorism.docx (D31266049)
final thesis_24-11-2018.docx (D44533842)
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6141335/
https://www.slideshare.net/Apollo_Hospitals/jaundice-a-brief-historical-perspective
https://www.researchgate.net/publication/333451016_Maternal_and_fetal_outcome_in_jaundice_complicating_pregnancy
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5559545/
https://www.aafp.org/afp/1999/0215/p829.html
https://fpnotebook.com/gi/Lvr/ActHpts.htm
https://www.babymed.com/newborn-first-year/jaundice-newborns
https://www.slideshare.net/imanswati/jaundice-in-pregnancy-64902486
718f4e7a-bf6a-4185-b4c6-4dd964a27387

Instances where selected sources appear:

27

PLAGIARSM CERTIFICATE

This is to certify that this dissertation work titled **"Jaundice Complicating Pregnancy – Maternal and Fetal Outcome"** of the candidate **Dr. SARANYA.G** with **Reg. No.221716014** for the award of M.S in the branch of **OBSTETRICS AND GYNAECOLOGY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows nine percentage of plagiarism in the dissertation (D58569164)

Signature and Seal of the Guide

PROF. DR .GEETHA MAHADEVAN, ,M.D.,D.G.O.,
Professor of Obstetris and Gynaecology,
Institute of Obstetrics and Gynecology,
Egmore, Chennai

Sl. No.	Name	Age	IP No.	CH	WOG	Symptoms	Durations	Signs	Complications	BS	BP	Protein	Sugar	Hb	Platelet	Serum Bilrubin (mg%)	SGPT IU/L	SAP (KA)	HbsAg	P.Smear	Widal	Laptoaplrosis	diagnosis	Mode of Delivery	Baby Details	Specific	
1	LAKSHMI	25	4880	G3P2L2	24	1,2,3,4	2 Days	1	1	+	+	-	-	7	2.2	8.4	395	500	-	-	-	-	1	Abortion	1		
2	GOVINDAMMA	25	4781	G3P2L2	40	1,2,3,4	9 Days	1	-	+	+	-	-	10	2.1	4.6	80	90	-	-	-	-	1	Normal	alive 2.9		
3	AMMU	29	2835	G3P2L2	38	1,6,7	1 MONTH	1.5	-	+	+	-	-	11	2.1	4.1	70	1150	-	-	-	-	3	Normal	alive 3		
4	LATHA	22	5986	PRIMI	32	1,2,3,4	5 DAYS	1	-	+	+	-	-	10.5	2.2	5.5	110	120	-	-	-	-	1	Normal	ALIVE 2.2		
5	UMAPATHI	20	7436	G2P1L1	32	1,2,3,4	6 DAYS	1	1	+	+	-	-	10	2.2	4.8	55	250	-	-	-	-	1	Normal	ALIVE 1.95		
6	JAQULINE	24	7822	G2P1L1	34	1,2,3,4	7 DAYS	1.2	-	+	+	-	-	10.5	2.2	12.8	89	250	-	-	-	-	1	Normal	ALIVE 1.95	N.D	
7	PADMAVATHY	25	8416	PRIMI	36	2,3,4	1 DAY	1	5	+	+	-	-	10	2.1	14	430	520	-	-	-	-	4	Heptic Coma - Death			
8	RANI	23	8050	PRIMI	34	-	3 MONTHS	1,2,3	-	+	+	-	-	10	2.2	2.4	60	80	-	-	-	-	1	Normal	Alive 1.7		
9	MANJERI	22	14388	G3P2L2	12	2	4 DAYS	1,3	1	+	+	-	-	7	2.2	2.6	200	215	-	-	-	-	1	Abortion			
10	AMSAVANI	25	11436	G4P2L2A1	34	2	8 DAYS	1	-	+	+	-	-	10	2.2	2.6	70	90	+	-	-	-	1(B)	Normal	Alive 1.95		
11	REVATHY	29	13380	PRIMI	24	2	12 DAYS	1	-	+	+	-	-	10.5	2.1	2.5	60	90	-	-	-	-	1	Normal	Alive 2.5		
12	LAKSHMI	23	12562	PRIMI	30	1,3,4	7 DAYS	1	-	+	+	-	-	10	2.1	14.4	60	100	-	-	-	-	1	Normal	Alive 1.1	N.D	
13	RISHVANA	27	13106	PRIMI	34	1,2	9 DAYS	1,3,4	1	+	+	-	-	8	2.1	4.4	90	100	+	-	-	-	1(B)	Normal	Alive 2.7	N.D	
14	ARUNAKUMARI	22	15635	G3P2L2	34	3.4.5	2 DAYS	1	2	+	+	+++	-	10	1.5	2.5	60	100	-	-	-	-	2	Normal	Alive 2.25		
15	MARY	21	15778	PRIMI	38	1,2,3,4,8	4 DAYS	1.2	3	+	+	-	-	10	2.1	16	430	300	+	-	-	-	1(B)	DIVC - Death			
16	SHANTHI	29	15057	G2P1L1	34	2	7 DAYS	1,2	1	+	+	-	-	8	2.2	2.5	300	310	-	-	-	-	1	Normal	Alive 2.5		
17	SUGUMARI	20	15334	PRIMI	34	2	8 DAYS	-	-	+	+	-	-	10	2.1	2.6	60	90	+	-	-	-	1(B)	Normal	Alive 2.25		
18	BHAVANI	23	18426	G2P1L1	40	3,4	9 Days	-	-	+	+	-	-	10	2.1	2.5	90	110	+	-	-	-	1(B)	Normal	Alive 2.75		

19	PRIYA	19	19164	G2P1L1	32	1,2,3,4	3 DAYS	1,2	1	+	+	-	-	6	2.2	14.2	320	350	-	-	-	-	1	Normal	Alive 1.4	N.D (PPH)	
20	SUJITHRA	20	20968	G3P2L2	12	4	7 DAYS	-	1	-	-	-	-	9	2.1	2.5	55	50	-	-	-	-	5	Normal	Alive 2.4		
21	RAVANAMA	27	31870	G2P1L1	28	1,3,4	10 DAYS	1,2	-	+	+	-	-	10	2.1	8.4	150	175	-	-	-	-	1	Normal	Alive 2.75		
22	ESWARI	35	23340	G3P2L2	6	1,2,3,4	3 DAYS	1	1	+	+	-	-	7	2.2	10.8	318	210	-	-	-	-	6	Total Abdominal Hysterectomy			
23	BALGASH	30	25475	G3P2L2	40	3,4	5 MONTHS	1,3,4	-	+	+	-	-	10	2	2.5	75	75	+	-	-	-	4	Normal	Alive 2.5		
24	SHANTHI	28	26746	G3P2L2	36	1,2,3,4	3 DAYS	1,2	-	+	+	-	-	10	2.3	4.8	85	100	-	-	-	-	1	Discharge on Request and Lost			
25	ARIYAMA	23	27043	G2P1L1	34	4,5	3 DAYS	-	2	-	-	+++	-	10	1.3	2.8	200	150	-	-	-	-	2	LSCS	Aive 2		
26	KAVITHA	22	31849	G2P1L1	26	1	9 Days	1,2	-	+	+	-	-	10	2.1	4.5	120	170	+	-	-	-	1	Normal	Alive 2.5		
27	MUTHULAKSHMI	29	33578	G3P1L1	32	3,4,5	3 DAYS	-	2	+	+	+++	-	10.5	1.5	2.6	75	80	-	-	-	-	2	LSCS	Alive 1.65 Alive 1.6		
28	DHANAM	19	2903	PRIMI	8	3,4	10 DAYS	1	5	+	+	-	-	10.5	2.1	14.7	197	110	+	-	-	-	1(B)	Abortion			
29	ADIYAM	36	3622	G2P1L1	32	1,	6 DAYS	1	-	+	+	-	-	11	2.2	3.2	65	70	-	-	-	-	1	Discharge on Request and Lost Follow up			
30	RENUKA	22	34227	PRIMI	12	-	2 Days	1	-	+	+	-	-	11	2.1	2.8	105	90	-	-	-	-	1	Discharge on Request and Lost Follow up			
31	LEEMA	26	2278	G2P1L1	22	1,3,4	4 DAYS	1	-	+	+	-	-	11	2.1	16	240	200	-	-	-	-	1	Normal	Still born 1		
32	LAKSHMI	30	2264	G3P2L2	34	3,4	3 DAYS	1	2	+	+	+++	-	10.5	1.4	2.8	140	120	-	-	-	-	2	LSCS	Alive 1	N.D	
33	BINDHU	22	4535	PRIMI	32	1,3,4	7 DAYS	1	-	+	+	-	-	10	2.1	13.6	94	100	-	-	-	-	1	Normal	Alive 2.1		
34	HASEENA	24	3423	G4P3L3	32	1,	7 DAYS	1	3	+	+	-	-	5	2	14	135	1526	-	-	-	-	7	Normal	Still born 1.8	PPH	
35	MARIYAMA	20	10032	G2P1L1	32	1	7 DAYS	1	4	+	+	-	-	11	2	16	145	1226	-	-	-	-	7	Normal	Still borh 1.5		

36	PUSHPAVALLI	20	10648	G2P1L1	32	1,2	3 DAYS	1	-	+	+	-	-	10	2.2	3.4	95	100	-	-	-	-	1	Discharge on Request and Lost Follow up			
37	SHANTHI	27	5803	G5P3L3A1	34	1,2,3,4	6 DAYS	1	1,2	+	+	-	-	7	1.4	12.8	220	315	-	-	-	-	2	Normal	Alive 2		
38	MEENA	21	5000	PRIMI	40	1,2	7 DAYS	1,2	-	+	+	-	-	10	2.1	4.4	160	170	-	-	-	-	1	Normal	Alive 2.7		
39	VARAOLAKSHMI	35	25653	PRIMI	32	1	1 YEAR	1,3,4	-	+	+	-	-	10	2.2	5.2	73	408	-	-	-	-	4	Normal	tillborn 1.5		
40	ASHA	28	25684	P3L3	-	1,3,4	7 DAYS	1,2	1	+	+	-	-	8	2.1	5.8	80	95	-	-	-	-	1	Normal	IUD 1.5	PPH	
41	SUMATHI	21	23407	G2P1L1	34	1,2,3,4	4 DAYS	1	1	+	+	-	-	5	2.1	17.2	180	100	-	-	-	-	1	Normal	Alive 2.4	PPH & DEATH	
42	KAMATCHI	27	8066	G3P2L2	32	1,2,3,4	6 DAYS	1	5	+	+	-	-	10.5	2.1	10	390	500	-	-	-	-	1	Normal	Still born 1	COMA DEATH	
43	JAYANTHI	22	33062	G2P1L1	12	1,2	7 DAYS	1	-	+	+	-	-	11	2.2	12	410	293	-	-	-	-	1	Abortion			
44	SUGUNA	22	31644	G2P1L1	28	1,2,3,4	7 DAYS	1,2	-	+	+	-	-	10	2.1	3.2	70	90	-	-	-	-	1	Discharge on Request and Lost Follow up			
45	MUNIYAMA	26	28342	PRIMI	38	1,3,4	1 MONTH	1	-	+	+	-	-	10	2.1	11.5	175	200	+	-	-	-	1(B)	Normal	Alive 2.7		
46	MANIMEGALAI	27	29056	G2P1L1	20	1,2,3,4	10 DAYS	1	1	+	+	-	-	7	2.1	6.6	90	100	+	-	-	-	1(B)	Discharge on Request and Lost Follow up			

47	UMAPATHI	21	30775	G2P1L1	28	2,3,4	3 DAYS	1	-	+	+	-	-	10	2.2	2.5	60	7	-	-	-	-	1	Discharge on Request and Lost Follow up			
48	DHANAMAL	26	11456	PRIMI	34	5	1 DAY	1	2	+	+	-	-	10	1.3	3.4	70	75	-	-	-	-	2	LSCS	Still born 1.5		
49	SUMATHI	22	29351	PRIMI	34	1,2,3,4	4 DAYS	1	1	+	+	+++	-	7	2.5	4	105	100	+	-	-	-	1(B)	Normal	Alive 1.75 Alive 1.65	PPH & DEATH	
50	SHANTHI	21	15778	G3P1L1A1	32	4,5	1` DAY	-	2,3	-	-	+++	-	10	1	2.7	150	160	-	-	-	-	2	LSCS	Still born 2.5	DIVC Death	
51	VANITHA	22	23127	PRIMI	40	1,2	5 DAYS	1	-	+	+	-	-	10	2.2	6.5	200	215	-	-	-	-	1	Discharge on Request and Lost Follow up			
52	CHINAPAPPA	24	11446	G2P1L1	36	1,2	1 DAY	1	1	+	+	-	-	7	2.2	3	60	90	-	-	-	-	1	Discharge on Request and Lost Follow up			
53	LATHA	23	11091	G2P1L1	30	1	6 MONTH	1,3,4	1	+	+	-	-	8	2.1	3.5	60	90	-	-	-	-	4	Discharge on Request and Lost Follow up			
54	GOWRI	23	33584	G4P3L2	32	1,2,3,4	6 DAYS	1,2,3	1,3,4	+	+	-	-	8	2.1	8.1	190	200	-	-	-	-	8	HRF AND DEATH			

55	SELVI	26	32617	G4P2L2A1	8	-	3 DAYS	1	1,3,4	-	-	-	-	4	2.1	4.6	91	120	-	-	-	-	9	HRF AND DEATH (Spontaneous expulsion)			
56	VASANTHA	23	11664	PRIMI	32	2,3,4	7 DAYS	1	-	+	+	-	-	10	2.2	2.9	60	70	+	-	-	-	1(B)	Discharge on Request and Lost Follow up			
57	BHAVANI	36	11780	G2P1L1	16	1,2,3,4	1 DAY	1	-	+	+	-	-	10	2.2	5.8	98	100	-	-	-	-	1	Discharge on Request and Lost Follow up			
58	IRMALA	19	12914	PRIMI	20	1,2,3,4	6 DAYS	1	-	+	+	-	-	10	2.1	4	70	77	-	-	-	-	1	Discharge on Request and Lost Follow up			
59	SHENMUGAVALLI	25	12823	PRIMI	32	1,2,3,4	8 DAYS	1,2	-	+	+	-	-	10	2	12.9	260	250	-	-	-	-	1	Discharge on Request and Lost Follow up			
60	ALAMELU	25	14087	G3P1L1A1	38	1,2,3,4	8 DAYS	1,2	-	+	+	-	-	10	2.1	8	270	350	-	-	-	-	1	Normal	Alive 2.8		
61	VANAJA	25	9289	PRIMI	32	2,3,4	7 DAYS	2	-	+	+	-	-	10	1.7	2.6	60	17	-	-	-	-	1	Normal	Still born 1.7		

62	VADIVU	21	22782	G3P2L2	40	2,3,4	6 DAYS	1	1	+	+	-	-	4	2.2	14.9	450	150	-	-	-	-	1	LSCS	Alive 2.5	PPH & DEATH	
63	AMSA	33	16472	G4P3L2	38	2,3,4	6 DAYS	2	-	+	+	-	-	10	2.1	2.5	172	90	+	-	-	-	1(B)	Normal	Alive 3		
64	SHANTHI	23	17838	PRIMI	32	2	5 DAYS	1	-	+	+	-	-	10	1.8	2.5	110	90	-	-	-	-	1	Discharge on Request and Lost Follow up			
65	SUMATHI	23	20554	G2P1L1	32	1,6	1 MONTH	1	-	+	+	-	-	10	1.9	14.2	160	1554	-	-	-	-	3	Normal	Alive 2.4		
66	VIJAYALAKSHMI	22	14337	PRIMI	38	1,2,3,4	4 DAYS	1	1,5	+	+	-	-	4	2.1	10	460	405	-	-	-	-	1	Forceps	Alive 2.7	COMA DEATH	
67	KAMALA	23	23143	PRIMI	32	1,2,3,4	6 DAYS	1	-	+	+	-	-	10	2	7.8	200	210	-	-	-	-	1	Normal	Still Born 2.5		
68	PUSHPA	25	29447	PRIMI	34	1,2,3,4	7 DAYS	1	-	+	+	-	-	10	2	10	200	115	-	-	-	-	1	Normal	Alive 2.5		
69	KAVITHA	22	29721	PRIMI	34	1,2,3,4	4 DAYS	1	-	+	+	-	-	10	1.9	4.3	80	100	-	-	-	-	1	Normal	Alive 1.4	N.D	
70	SHANTHI	21	17952	G2P1L1	40	1,2,3,4	5 DAYS	1,2	1	+	+	-	-	4	2.1	15	405	450	-	-	-	-	1	Normal	Alive 4	PPH & DEATH	
71	VEERAMA	25	26029	PRIMI	34	1,2	10 DAYS	1	-	+	+	-	-	10	2.1	4.8	80	90	-	-	-	-	1	Normal	Alive 2.5		
72	KANNAGI	27	27119	PRIMI	32	1,2,3,4	6 DAYS	1,2	-	+	+	-	-	10	2	6.8	110	100	-	-	-	-	1	Normal	Alive 1.75	N.D	
73	MALA	20	14335	PRIMI	32	1,2	7 DAYS	1	-	+	+	-	-	10	2.1	8	90	115	-	-	-	-	1	Normal	Alive 1.3	N.D	
74	JAMUNA	20	27221	G2P1L1	32	1,2	7 DAYS	1	-	+	+	-	-	10.5	2.1	9	150	200	-	-	-	-	1	Discharge on Request and Lost Follow up			
75	AMUDHA	20	29891	PRIMI	32	2	3 DAYS	-	-	+	+	-	-	11	2.5	2.5	60	70	-	-	-	-	1	Discharge on Request and Lost Follow up			
76	KALA	22	17600	PRIMI	36	1,7	1 MONTH	1	-	+	+	-	-	10	2.1	6.7	115	1650	-	-	-	-	3	Normal	Alive 2.2		

77	HEMA	23	17541	PRIMI	10	1	6 DAYS	1	-	+	+	-	-	10	2	6	95	110	-	-	-	-	1	Abortion			
78	KANNIYAMMA	21	37270	G2P1L1	20	2	5 DAYS	1,2	-	+	+	-	-	11	1.9	3	90	100	-	-	-	+	8	Discharge on Request and Lost Follow up			
79	USHA	18	31432	PRIMI	30	1	7 DAYS	1	-	+	+	-	-	10.5	2	5	100	90	-	-	-	-	1	Normal	Alive 2.5		